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ORIGINAL COMMUNICATIONS.

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CHRONIC DEAFNESS—AN ENDOCRINE STUDY OF A THOUSAND CASE HISTORIES.*†

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The subject of subacute and chronic nonsuppurative disease of the middle ear is one to which much attention has been given of late years, but at present the results, although considerable, have not been as correspondingly great as have those in the case of chronic suppuration. The most striking headway has been made in the recognition of the causes leading to chronic deafness and great advances have resulted in the treatment of those conditions of the upper air passages which are in part responsible for it.

One of the greatest difficulties in the way of investigation has been the lack of pathological material, especially in the early stages, wherewith to differentiate the various conditions met with. Persons suffering from chronic deafness due to nonsuppurative middle ear changes do not die from their disease; whilst, should they succumb to other maladies during the early stages of their ear affections, it is very possible that it has not been diagnosed, and the chance of a competent pathologist and otologist obtaining their temporal bones is unlikely.

There is great diversity in the classification of these forms of deafness, and the student often finds that a diligent perusal of their various descriptions only serves to render him the more bewildered. In systematic teaching, it is a matter of some difficulty to put forward a good arrangement, clear and intelligent to the learner. The

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one which I believe to be correct is to recognize only two main forms, catarrhal inflammation and otosclerosis. Of these, the first is the more common and, as the name implies, is inflammatory in type. It is, moreover, a true middle ear condition, whereas the second—otosclerosis—is neither inflammatory nor tympanic, but is a state wholly or in large measure affecting the bony labyrinthine capsule. Confusion has arisen between these two forms, because, in the first place, as regards symptoms, there is a good deal of similarity between the later stages of catarrhal middle ear inflammation and otosclerosis; and in the second place, the two conditions may be coexistent in the same persons.

The secret of the successful treatment of catarrhal otitis lies in the correction of abnormal conditions of the upper air passages. Another important factor in its management lies in the combating of these conditions at as early a stage as possible. Unfortunately, there is no organ so persistently neglected by the laity as the ear. A man who is the subject of an affection of the eye does not usually wait until his sight is irretrievably impaired before he seeks advice, but persons who are gradually failing in hearing will wait until their deafness is far advanced before they even contemplate investigation and treatment. This neglect handicaps the otologist most severely in his work, and helps to bring the science of otology into disrepute.

At first sight there appears to be little in common between inborn derangements of function and structural defects, but on further consideration the difference is seen to be apparent, rather than real. Almost any structural defect will entail some disorder of function; sometimes the former is almost inappreciable, and the resulting functional disorder so conspicuous that it completely overshadows the defect to which it is due. Very slight structural changes may lead to profound functional derangements, as witness the effects of atrophy of the thyroid gland, whether congenital or acquired in later life, and the stormy metabolic disorders which may ensue upon comparatively insignificant morbid changes in the pancreas. Thus, briefly, I wish to bring your attention to a large series studied intimately from this angle.

THE THYROID.

Any account of the endocrine glands is bound to begin with the thyroid. This is because it has been studied longer and more intimately than the others, with the exception of the pancreas, and research into its functions has in no small measure opened the way to comprehension of the rest.

The reasons for this prominence are obvious. In the first place, enlargements of the organ are very common and, owing to its super-

ficial position in the neck, such enlargements are easily visible and, if considerable, are very unsightly. Further, the fact that goitre is endemic in certain districts all over the world, early set men's thoughts at work to find a cause for this phenomenon. Later it was shown by Graves and Basedow that certain forms of goitre, even though the enlargements were slight, were associated with very serious symptoms, prominence of the eyeballs, rapid pulse and breathing, nervous imbalance and progressive weakness.

Associated with goitre in certain cases was found the condition of "cretinism", a lamentable disease in which both bodily and mental development is arrested at an early age, and the individual becomes a more or less idiotic and deformed dwarf, with a strange appearance of old age in childhood. We have, therefore, simple goitre, or enlarged thyroid, causing no general symptoms; goitre associated with cretinism in some cases and with Graves' disease in others; further, both cretinism and Graves' disease arising without apparent enlargement of the thyroid; and finally, thyroid failure, with and without myxedema, associated apparently with all stages of hypertrophy and atrophy of the gland. All these different manifestations of disturbed function become quite comprehensible when we realize that they depend essentially on an excess, deficiency or abnormality of the internal secretion of the gland, the size of which is no certain index of its activity.

Thus, the gland may be enlarged without disturbance of its function, or to compensate for a shortage in the system of a certain element, thus giving rise to simple goitre; it may be enlarged and coupled with excessive secretion (Graves' disease), equally with secretory failure causing destruction of its function (goitrous cretinism). Further, there may be excess of function without enlargement, now generally called hyperthyroidism, or, conversely, a diminished function without enlargement or apparent atrophy, causing hypothyroidism.

In a series of over 1,000 consecutive case histories that I have studied, 148, or 15 per cent, presented thyroid disease, either hyperor hypofunctional in character. In this series of cases, deafness was a presenting symptom in 18 per cent, and of this number, over 80 per cent fell in the hypofunctional group. Two common symptoms seen in deaf people, namely, tinnitus and vertigo, were noted in the histories in this series as 34 per cent and 15 per cent, respectively. The predominance of tinnitus was possibly due to the fact that it was observed in all cases in which secondary anemia was a symptom.

I cite the following two thyroid cases as illustrating very well the many points cited above.

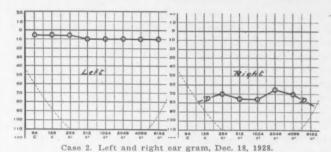
Case 1: Male, age 16 years, freshman in college, presented himself with disturbing tinnitus in both ears, which was intermittent and increased when the hearing was less acute. Both ear canals were very scaly and dry, and he felt that he was having an increased difficulty in understanding conversation. There had been increasing ear trouble since he was 2 years old, when he had his first attack of pneumonia, associated with bilateral incisions of the drum membranes. For the next few years he had repeated attacks of earache, which were relieved by incisions of the drum, and also several attacks of pneumonia. In addition, he had had measles, two tonsillectomies, and at one time several short attacks of blurring of vision. In the main, he had been strong and active. He had an attack of hives last spring. The residual history not informative.

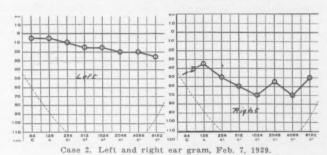
Physical examination showed good development and fair nourishment. There was slight dandruff, thick, grayish skin, an exudate from both canals, the skin of which was slightly red and the canals markedly narrowed. The eyes were negative. The nose showed inflamed mucosa and crusting. The abdomen was rigid over right upper quadrant, especially near the midline. The remaining examination was not remarkable. The urine volume was normal, with good elimination, and fair balance. The protein intake was adequate: the nitrogen partition, normal; one specimen showed albumin; the sediments were negative. The blood morphology showed a definite lymphocytosis, the chemistry was normal; the serologicals were negative. The phthalein test was slightly below normal. The galactose tolerance was definitely depressed. The patient was 10 per cent underweight, but showed a normal lung volume. Two confirmatory basal tests gave an average rate of -18 per cent, coupled with a slightly slow pulse. An X-ray of the sinuses and mastoids was negative. The eyes were normal except for marked cutting of the upper fields due to lid droop. A heart examination showed a normal organ. An audiogram indicated depression throughout the auditory curve. A test of liver function by the duodenal method (McClure) indicated a considerable disturbance in biliary function. The Graham test was positive.

Diagnosis: Hypothyroidism (mild) with liver dysfunction.

Comment: The pathology in this case is at first sight not clearly defined. The low basal rate, a low sugar tolerance and lymphoid blood would be consistent with a pituitary dysfunction, but none of the other findings are confirmatory. That he was above the normal, or better, the average height, at the age of 16 years, might also be adduced to support a pituitary hypothesis. The results, however, of

the Graham test together with that of the duodenal function study showed definitely that there was a liver dysfunction. This finding would explain those observations inconsistent with a thyroid failure which the major portion of the test suggested. Association of deranged liver function with thyroid disease (hypofunctional) is a fairly common finding. The diagnosis as given above was offered. The local treatment consisted in keeping all water from the ear canals, the skin and exudate mopped clean, care being taken not to break the underlying canal skin. Small daily doses of thyroid with





two of his meals, increased to the three meals, was initiated and in two weeks he began to show a marked change for the better. This improvement has increased with the realization of the necessity for constant medication.

Case 2: A married man, age 56 years, head of an industrial organization, complained of severe frontal headaches, from which he had suffered for the past 10 years. These have been associated with attacks of vertigo, during some of which the patient has fallen. Twenty years ago, he developed tinnitus in the right ear for a short time and this recurred 10 years later and was accompanied by nausea

and vomiting. The attacks recurred at two or three-month intervals and were associated with progressive loss of hearing in the right ear. Three years ago, the tinnitus ceased, as did the nausea and vomiting. The severe frontal headaches have persisted, manifesting themselves after the patient had gotten at work, even though he had felt perfectly well up to that time. A few minutes' rest in a horizontal position relieved all symptoms. If the attack occurred while walking, he experienced some vertigo and a sense of insecurity. He might fall, but recovered in a few minutes. There was no frothing at the mouth or loss of sphincter control. His family history does not seem to be relevant. He had been married for 21 years, his wife had one miscarriage during the first year of the marriage and had never conceived again, although contraception has not been practiced. He recorded several minor ailments: scarlet fever, quinsy, sore throats, a blow to the head at the age of 7 years, with tenderness persisting for a year. At the age of 21 years he was thrown from a carriage, breaking two teeth. His remaining history showed slight dyspnea on exertion, constipation of 40 years' duration, though for the past few days the bowels have relaxed with warm weather. There was a slight ataxia.

Physical examination showed an obese man, with florid complexion, some dilatation of the scleral vessels, the left ear was normal, while the right showed a retracted drum, and the audiogram indicated marked bilateral depression of aural acuity. There was some cyanosis of lips, the teeth showed much dentistry; the heart and lungs were apparently negative. There was a thick pad of abdominal fat. He had some clubbing of the distal interphalngeal joints of all fingers; there were two areas of skin pigmentation over right tibia and three on left. The knee jerks were hyperactive. The urine volume was distinctly above the normal; elimination was high and the balance very poor. His protein intake was entirely inadequate, but the partition formula was normal. A small amount of albumin was reported. The blood morphology was substantially normal, with the exception of a 3 per cent eosinophilia. The blood chemistry showed a high uric acid. The serological tests, including the results of a spinal puncture, were normal. Both phthalein and CO2 were normal. The patient was 38 per cent overweight and appreciably below his predicted lung volume. The basal rate was -31 per cent, with normal physical findings. X-ray showed prominent diploic veins and a retained root which was not infected, while a gastric series showed a mass in the right iliac fossa, the stomach and duodenum being negative. The neurological examination was suggestive of a

labyrinthitis with partial destruction of the end-organ. The eye examination showed a moderate vascular sclerosis, yellowish discs, and slightly hazy lenses, but was otherwise substantially normal. The Barany test entirely eliminated a pontine tumor, leaving the suggestion only of an infiltrating edema of the eighth nerve. The galactose test was normal; he reacted positively to a number of proteins by the endermal method.

Diagnosis: Hypothyroidism.

Comments: A number of findings in this examination could be interpreted in terms of a primary or secondary pituitary involvement. The low basal rate was not inconsistent with a pituitary condition, as the patient's low protein intake would exercise a definite downward influence. There were several basal rate measurements, all of which showed a definite downward tendency. The normal galactose test practically eliminated the pituitary, though not ruling out a central nervous lesion. Other observations, including the normal lumbar puncture with normal spinal fluid, rendered this latter improbable. A thyroid failure was the only condition consistent with all of the observations. On this basis, treatment was instituted, consisting of carefully controlled thyroid medication. The patient has shown very marked and steady improvement, which undoubtedly will continue.

THE PITUITARY.

In the case of the pituitary body the intimacy of connection with the circulatory and nervous systems is not so marked as in the cases of the thyroid and suprarenal glands. The small size of this organ and its proportional blood supply suggest that its internal secretions must be of exceptional potency to exercise the powerful effects which they certainly do. The vital importance of the pituitary body is, however, suggested by its anatomical relations, for nature has taken altogether exceptional precautions to protect it from injury (as with the labyrinth of the ear).

The gland, however, although thus protected from injury and difficult of access for operation or experiment, is not immune to disease. Thus, as deficiency in the thyroid function will produce one set of conditions (cretinism) in infancy or childhood, and quite a different syndrome (myxedema) in adults, so an excessive or prolonged over-activity of the pituitary function during the growing years may produce a giant, while a similar hyperfunction in later life, after the period of normal growth, will produce the deformities incident to acromegaly.

Of no less importance are the functions of the posterior portion, which, it will be remembered, is a direct derivitive of the brain itself, and thus has certain affinities with the suprarenal body, as the anterior portion has with the thyroid. This portion is concerned with the metabolism of sugar. Also of importance is its association with the circulation, and therefore of blood pressure. Diminished activity of the gland is associated with torpor as well as adiposity, while dysfunction of the pituitary gland has a profound influence on the mental and emotional state.

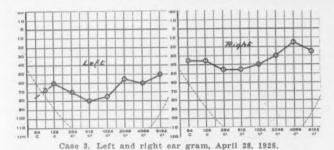
Again, in this same series mentioned above, 289 cases, or 29 per cent, showed frank pituitary involvement. In this series of cases deafness was a presenting symptom in 22 per cent, and of this group, 75 per cent presented the dysfunctional type of pituitary disorder. Again, taking tinnitus and vertigo as frequent accompanying symptoms in deaf individuals, the above symptoms presented themselves in a ratio of 36 per cent and 24 per cent, respectively. As in deafness, so with the presenting symptoms of tinnitus and vertigo, dysfunction of the gland predominated, the anterior lobe being generally hypo-, the posterior, hyperactive.

As illustrative cases, the following two bring out the suggestive points in question:

Case 3: A single woman, telephone operator, age 28 years, presented as her chief complaint an intense, deafening tinnitus in both ears which was of fours years' constant duration. Of late the intensity had markedly increased, while for the past two years the hearing of the left ear had rapidly decreased, the hearing of the right ear having diminished distinctly two years earlier, or four years ago. The immediate family history was not available. The patient recorded the usual minor ailments, scarlet fever, influenza in 1918 and a tonsil and adenoid operation last year. She had an abscess in the right ear two years ago, with spontaneous discharge, and had been subject to severe colds in the head, with considerable nasal discharge, for a very long time. She had been troubled earlier with flatulency and had a tendency to constipation. The menstrual history was substantially negative.

The physical examination showed a marked degree of obesity, which was said to be uniformly distributed, an impression not borne out by the measurements, which showed a girdle type. There was slight pilosity about the areolae, tendency to masculine distribution of pubic hair, and some pilosity on chin. She showed an eczema involving both legs. The urine volume was normal, the elimination poor, the balance fair; indican was increased. The patient's protein

intake was definitely below a maintenance level, and the residual nitrogen showed an upward tendency. The blood morphology was substantially, and the blood chemistry completely normal. The serologicals were negative; phthalein and CO₂, normal. The patient was 59 per cent overweight and as the sitting height index was well above the normal, her obesity was even more pronounced. The lung volume was slightly reduced. The first basal rate showed —11 per cent, the second —19 per cent, with substantially normal physical findings. X-ray of the skull, sella and sinuses were negative, as was

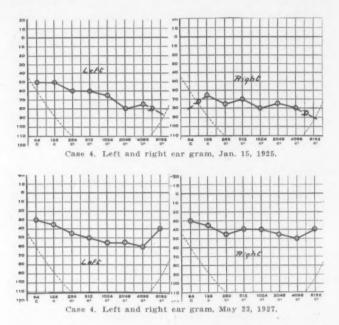


Case 3. Left and right ear gram, Dec. 11, 1928.

a nose and throat examination. The Barany test showed sluggish reaction time, while the audiogram gave evidence of lowered aural acuity. This finding, coupled with bone conduction tests and other measurements, indicated an otosclerosis. The eye examination showed vascular hypertension and some enlargement of the right blind spot. A duodenal function test (McClure) showed normal biliary function; the sugar tolerance was somewhat lowered, she being positive with 30 gm.

Diagnosis: Pituitary dysfunction (anterior lobe hoppo-, posterior lobe hyperactive).

Comments: The basal metabolic rate is lowered, with but slightly lowered blood pressure, the urine examination indicates a low protein intake, with a raised residual nitrogen fraction. The blood uric acid is above the normal, the galactose tolerance reduced. There was secondary anemia, with a lymphoid blood. The treatment has consisted of attention to the intestines and the daily administration of 20 gr. of anterior lobe pituitary extract under careful supervision. There has been a loss of the excessive weight and a very decided diminution in the tinnitus, as well as a distinct improvement in the hearing, especially the left ear.



Case 4: A married woman, age 30 years, gave as a chief complaint an increasing deafness in both ears. This condition apparently followed several attacks of sore throat, the first of which occurred eight years ago. A tonsillectomy a year later did not seem to have exercised any influence. Two maternal uncles, as well as a brother, showed similar deafness. The patient had the diseases of childhood, a chronic bronchitis, an appendectomy in 1923, and later a laparotomy for some reason, to her unknown. The patient suffered now

from marked dizzy spells and was easily fatiguable. The menstrual history showed an onset at 16 years and was said to be regular.

Physical examination showed an obstruction in nasal breathing and two missing teeth, otherwise the findings were quite normal, except for the deafness. The urine showed a normal volume, elimination decidedly poor and balance poor. One specimen showed albumin, both sediments being negative. The residual nitrogen fraction in one specimen was distinctly high. Phthalein was normal. The serologicals, negative. The galactose test was positive with 20 gm., a definitely lowered tolerance. The blood morphology showed the hemoglobin and red count slightly low; the blood was markedly lymphoid in character. The blood chemistry showed a high uric acid. The patient was 8 per cent overweight and exhibited but threefourths of her predicted lung capacity. The basal metabolic rate was -17 per cent, with lowered temperature and blood pressure. The eyes showed yellowish discs, somewhat enlarged blind spots, with definitely contracted color fields. The X-rays and neurological examinations were negative. The Barany test showed sluggish responses, while the ears were quite typical of otosclerosis.

Diagnosis: Pituitary dysfunction (anterior lobe under-, posterior

lobe overactive).

Comments: This case showed very marked psychotic changes, a phase which I find many otosclerotics show to a high degree. Under large doses daily of anterior lobe pituitary extract the mind has cleared decidedly, the hearing improved markedly. I might add that the hearing improved more in the first six months of active medication than it did during the next year, but during the past few months it again has started to improve, perhaps because the dose has been raised to nearly double the primary amount.

THE OVARIES.

Without going into the physiology of the ovaries it is sufficient to say that there can be very little doubt that these organs play a vital part in the production of secretions, which, although we are ignorant of their full importance, are necessary for the development of a normal individual; not only insofar as sexual characteristics are concerned but even with regard to the adequate working of the body metabolism.

In the current series of cases studied, 119, or 12 per cent, were ovarian in origin and all of these exhibited failure, either of functional or surgical origin. Deafness was a presenting symptom in 13 per cent, while 34 per cent showed tinnitus and 22 per cent, vertigo.

As an illustrative case I cite the following as rather typical:

Case 5: A married woman, age 24 years, had as a presenting symptom an increasing binaural deafness, with vertigo and tinnitus which kept her awake at night. There was a history of deafness in the family and that of the patient was first noted with the establishment of the catamenia. When the tinnitus was less intense the hearing was somewhat clearer. She was able to hear over the telephone very easily and she had no difficulty in hearing music. The vertigo was more marked when her ever-present constipation was more pronounced. She noted the usual children's diseases and influenza in 1919.

The physical examination showed a very normal individual, except for the ear condition. The urine showed a lessened amount with a trace of sugar present, as well as an occasional cast and many calcium oxalate crystals. The residual nitrogen fraction was markedly raised. The blood morphology showed a marked lymphocytosis, with a slight increase of leukocytes. The blood chemistry showed a blood sugar of 125 m.gm., the level due probably to an emotional response to venipuncture. The serologicals were negative. Phthalein was a low normal; the CO₂, normal. The basal metabolic rate was —15 per cent, with a depressed temperature and pulse rate, and a blood pressure of 110/68 m.m. The eyes were normal. The Barany test was normal. The hearing acuity was depressed in both ears, but not equally; the bone conduction was greatly prolonged. The X-ray and neurological examination were entirely normal.

Diagnosis: Hypogonadism.

Comments: The picture in the above case is very clear-cut, but success in treatment is quite another story. For a period of several months I watched the result very closely of the active medication upon her ears, but beyond controlling the tinnitus I was unable to improve the hearing curve, although the patient physically felt very much less fatigued and accomplished her work with greater ease.

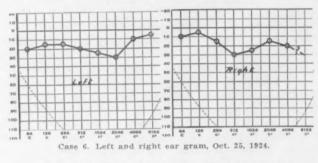
THE NONENDOCRINE GROUP.

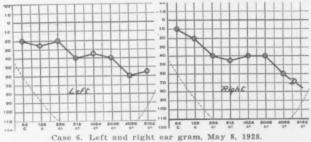
In this series of 1,000 cases, 415, or 42 per cent, initially presented stigmata suggesting an endocrine pathology, but failed on analysis to give evidence of endocrine disease and did demonstrate pathology of a nonendocrine character. The diagnosis as established covers most of the ills to which the body falls heir. Deafness as a presenting symptom occurred in 19 per cent, with tinnitus in 31 per cent, and vertigo in 20 per cent of the group. Deafness occurred more frequently in the cardiorenal group, tinnitus in the group of

the neuroses and psychoses, while vertigo predominated in that of lesions of the central nervous systems.

The following cases mark well the several points in the thesis:

Case 6: A single woman, age 65 years, who followed a professional activity, lecturing at times and traveling extensively at other times, during the past year-and-a-half complained of attacks of deafness and tinnitus following fatigue. She would become rested, but the deafness would persist. The deafness included the voice and music. Following each one of these attacks, which would last between one and three weeks, the recovery would not be quite com-





plete, thus leaving a residuum of deafness, but no tinnitus. Beyond the slowing up normal to a woman of her age, her past history was quite negative.

The physical examination was essentially negative beyond the deafness which has already been mentioned, with the exception of a slight metacarpal keratosis. The urine examination presented no point of serious abnormality; the occurrence of a rare hyaline cast was noted and a decidedly low value for the urea. This last could be ascribed to a dietary regulation, rather than to any kidney insufficiency. The

total nitrogen was low, while the residual nitrogen was high. The blood morphology was negative; the blood chemistry was negative, except that the urea nitrogen was slightly high. The serological tests were negative. Phthalein was normal; the CO₂ showing a slightly lower value. The basal metabolic rate, temperature and pulse rate were all within normal limits, the blood pressure being 138/86 m.m. The eye, Barany and neurological examinations were all negative. An X-ray series of the gastric tract showed poor elimination from the descending colon, but no growth; the fecal examination showing extensive putrefaction and large numbers of bacillus coli. She reacted positively to a number of proteins by the endermal method.

Diagnosis: Catarrhal deafness of toxic origin.

Comments: The entire picture presented an extremely well preserved woman of 65 years of age. Following the ingestion of the barium meal it was noted within 48 hours that the deafness had become less acute; as a result of this suggestion, the putrefactive changes were decreased by active measures and the tinnitus ceased while the hearing acuity improved. At the same time the foods to which she showed sensitivity were removed from her diet. On several occasions with a return of the deafness, another X-ray series would show a repetition of the above condition, following which the above treatment would give the desired relief.

Case 7: A married man, age 20 years, presented as a chief complaint an increasing bilateral deafness without vertigo for the past four years, but early in the deafness a very constant, distressing tinnitus appeared. During this past four years there was a period of nine months when the deafness remained stationary, then progressed again without any discernible reason. He complained of paracusis and improved hearing during amplification of sound. Beyond the father's deafness, the family history is negative. Married one year, wife never pregnant. Diphtheria and scarlet fever in childhood, typhoid fever with chorea following it 12 years ago, and influenza eight years ago. History otherwise was negative.

The physical examination show a slightly palpable thyroid gland and some acne on the face, but was otherwise insignificant. Beyond a small volume the urine was negative. There was a high residual nitrogen. The blood morphology showed a leukoid blood and a low color index; the blood chemistry was normal. The serological tests were negative. Phthalein, CO₂ and the vital capacity were normal. The basal metabolic rate was —3 per cent, with normal clinical findings, except of a slightly febrile temperature. The audiogram showed a material impairment of hearing; the bone conduction was

lengthened, the Barany test was normal. The eye examination showed slightly overfilled veins and marked contraction of form and color fields. The X-ray and neurological examinations were negative.

Diagnosis: Otosclerosis.

Comments: There is nothing in this examination to indicate any endocrine element. In fact, the whole examination is negative of any really significant departure from the normal. He was advised to change his work to a noisier place. He did this and found that the deafness bothered him less and he was less fatigued at the end of the day.

Case 8: A widower, age 46 years, father of the above case, who was said to be an otosclerotic. However, the deafness began 10 years ago following a severe head injury, and at the same time the vision became affected. The deafness had not perceptibly changed since the accident, although he has paracusis, improvement in hearing during amplification, but not tinnitus. The family history was not informative. The patient's marital history was negative and his own history, beyond bad teeth, which might well be charged to his severe accident, two or three fractures in childhood, and a recent transitory edema of the right ankle, was not remarkable.

The physical examination showed no evidence of an injury to the face; slight nasal obstruction without apparent blocking and was otherwise negative. The urine was normal in volume and elimination. Urobilinogen was reported as positive and the sediment showed numerous leukocytes and a rare, fine granular cast. The nitrogen partition showed a somewhat high residual fraction. The blood morphology showed a lymphocytosis and a 3 per cent eosinophilia. The blood chemistry was normal. The serological tests were negative. Phthalein and CO₂ were normal. The basal metabolic rate was +10 per cent, with a slightly rapid pulse rate and a lowered blood pressure. The eye examination showed an apparent marked contraction of form and color fields and a slightly hazy medium in the left eye. The audiogram showed a marked loss of hearing throughout, except in the upper part of the speech area. The Barany test showed depressed reaction time only, while the neurological tests showed nothing remarkable. The X-ray showed a small sella, but was otherwise negative. On aural inspection both drums were retracted, scarred, dry, and the bone conduction was positive to the Rinné test, the Weber equalizing to both ears,

Diagnosis: Chronic deafness (traumatic); possibly liver dysfunction.

Comments: While one or two points in this examination suggest a pituitary lesion and there is undoubtedly some pathology of the kidneys, there is no real evidence of any endocrine disturbance. The deafness was probably the result of the severe injury, as was the condition of the eyes. It is not remarkable that the X-ray did not show old fractures of the skull. Besides the kidney lesion, he showed a possible liver dysfunction.

These last two cases are cited as they were originally examined for a possible otosclerosis exhibiting the influence of heredity. The examinations, however, did not confirm this. The younger man was seemingly a true otosclerotic. The father, on the other hand, had a history of earlier trauma to the head, which could well be a direct factor in his deafness. Beyond the deafness they showed one other point in common which was most striking, namely, a marked and peculiar contraction of the form and color fields. This latter condition has been noted by one of my associates in two conditions in which a resultant toxemia is conceivably a factor, namely, pulmonary tuberculosis and gall bladder disease (unpublished data, A.W.R.). The older man gave certain evidences suggesting a possible liver dysfunction. It was impossible to complete the study with them and resolve this matter. One may speculate, however, on the possible existence of a dysfunctional liver state in both father and son, which might be conceived to influence their hearing acuity.

SUMMARY.

Patients exhibiting thyroid failure are prone to a marked fatiguability which develops with a less than normal expenditure of energy. In this way they resemble the neurasthenic, just as in their somnolency after food, they remind the observer of the lithemic subject. The individual is never fit; he is generally tired and finds work a burden; his concentration is never at its best when faced with a problem; in a word, he is as sluggish mentally as he is languid physically. The reflexes are tardy, but they are always present. Giddiness is yet another symptom which almost always presents itself at some stage of the disease and with which in some cases is associated a swimming sensation. Tinnitus aurium is common in the form of buzzing, roaring, whistling or shrieking noises; while such patients sometimes experience even more serious sensory disturbances, as the hearing of voices or the ringing of bells.

Myxedema is a frequent finding in thyroid failure, although an amyxedemic type has been described by this institution. The thickening involves the skin and, according to many observers, other tissues as well. I feel that it is not illogical to explain some of the deafness seen in these cases to a colloidal infiltration. Certainly, treatment with the correct thyroid substance reduces the edema and causes local improvement in the hearing.

Diseases of the pituitary gland present symptoms which are markedly influenced by the time of the appearance of the deficiency in relation to the status of maturity of the individual. The pictures of pre- and post-adolescent failure are too well known to require elaboration. With the pituitary as with the thyroid, suitable medication is extremely effective in those cases where careful laboratory and clinical studies have established a definite diagnosis. It is perhaps not too much to say that where organotherapy is administered after the definite establishment of the diagnosis, the use is less speculative than with certain of the simple inorganic remedies, which, in ameliorating one symptom are prone to initiate others. It is a matter of record, further, that in the endocrinopathies a whole host of unusual and even bizarre symptoms may develop which are seemingly wholly unrelated to the primary condition. Correction of the latter, however, by suitable therapy concomitantly rectifies many of the physical and mental deviations which have apparently existed coincidentally with it.

It is axiomatic that in every well defined disease condition, individual sufferers betray a tendency to variation from the type, which presumably expresses the personal idiosyncrasy, and presumptively arises from minor individual deviations of which today we have no knowledge. Just as in a group of so-called normal people, few, if any, will conform exactly to the average, so in disease the textbook picture is but seldom realized. Where a correlation exists between lowered basal metabolic rates and chronic deafness, it seems most probable that both are results rather than causes. The former indicates a lowered functional activity, a depression of the respiratory exchange, a diminished energic output. Similarly, in patients demonstrating a lowered aural acuity which cannot be traced to a demonstrable infective process, there are likewise observed evidences of a decreased metabolic level. The endocrine glands are among the powerful regulators of the levels of body metabolism. In conditions of functional failure, lowered basal rates are invariably found. Where these are associated with a loss of aural acuity, for which no infective or other direct or mechanical cause can be demonstrated. it has seemed a fair presumption that the hearing failure might equally be a result of lowered metabolism deriving indirectly from the endocrine condition. Experience has shown this to be the case in a very appreciable percentage of true otosclerotics. It follows as a corollary to this postulate that other and nonendocrine agencies producing lowered metabolic levels may equally affect the acuity of hearing as one indirect end-result. It is on this basis that we have sought

to explain those otosclerotics in whom no endocrinopathy was demonstrable.

Otosclerosis is not an inflammatory condition. It is a degenerative process in the organ of hearing as a whole and for this condition to result, seemingly a tendency toward that process must be present in the individual. This does not necessarily mean that hereditary tendency applies in every case. Again, the findings in each bone are not always the same, still there probably is an unrecognized ratio between old bone absorption and new bone deposition in its place. It would seem, therefore, necessary to study completely, not only the temporal bone, but also the brain, including the auditory nerve tract to the brain cortex. Many feel that when they hear the word otosclerosis, it is quite like a death warrant. Now, I think that the rate of progress in many otosclerotics is very slow; in fact, in many of them the process does not seem to change perceptibly from year to year. Primarily, why in one case embryonic maldevelopments should be represented by an overgrowth, whilst in other cases no such tendency occurs is as little known as it the cause of the development of tumors generally, and is at present completely unexplained. We feel, however, that one logical explanation can derive from the influence of the endocrine secretions on certain phases of metabolism. We feel that otosclerosis should be regarded as hyperplasia, since the reason why some cases of this disease do well under treatment is explained by the law of reversible reaction. In other words, an equilibrium which has been notably displaced toward one final state, is moved back toward the antithetical condition and deposition is followed by resolution.

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INTRACRANIAL COMPLICATIONS FOLLOWING SPHENOID INFECTION.*

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The subject of the intracranial complications of sphenoid infection is presented because of the great difficulties in the diagnosis and the therapy of these cases. They are apparently rare in occurrence, less than 100 having been reported up to the present, but five of the cases reported in this paper were seen in the past year in Mt. Sinai Hospital. The disparity in these figures is probably due to lack of complete post-mortem examinations and to the fact that the condition is usually unsuspected during the life of the patient. In studying the records of these cases one finds that a number of rhinologists did not diagnose the involvement of the sphenoid but the neurologists frequently suspected it. The diagnosis is at times extraordinarily difficult and, with all our present day diagnostic facilities, occasionally an impossibility. Systematic therapy, comparable to that of the intracranial complications of otitic or frontal sinus origin, is unknown. The technical procedures at our disposal today are totally inadequate in combating the intracranial lesions following sphenoid bone infections. The problems will probably become more apparent when the protocols of the cases are studied and for that reason I shall first cite the case histories and leave the discussion of them till later. The sphenoid infections presented here are those originating in the sphenoid bone, whether they arise in the sphenoid sinus or in the body of the sphenoid, as in one case. Those with cranial postmortem examinations, six in number, are presented first and then three with no post-mortem examinations but in whom the diagnosis seems definitely established by clinical and operative findings.

Case 1: E. B., female, age 47 years, admitted to the Eye Service on Sept. 12, 1923, with a diagnosis of glaucoma, because of frontal headaches for one year's duration, for which she had received eyeglasses. Ten days before admission she fell out of a canoe and three days later she had severe frontal headache and pain behind the eyeballs. She became progressively worse; occipital pain appeared, she vomited one day before admission, and could not recognize features of family.

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Physical Examination: Emaciated, poorly nourished, with anxious expression, left homonymous hemianopsia, rigid neck, slight Kernig, suspicion right Babinski, active delirium. Provisional diagnosis, meningitis possibly from sphenoid. Spinal tap showed turbid fluid under increased pressure, many thousand polynuclear leukocytes, Gram positive, short-chained cocci. Sept. 14, 1923: Signs more marked. Blood count: W.B.C. 23,100, polys. 83 per cent. Nose examination by a member of the staff reported negative. Sept. 15: Ear examination negative. X-ray of sinuses negative. Temperature 99° to 100°, respiration 20 to 24. Urine negative. Blood Wassermann and blood culture negative. Sept. 16: Exitus. Post-mortem examination revealed a basilar meningitis, purulent encephalitis, large sphenoid containing greenish mucoid pus—all showed Gram positive, short-chained cocci.

This case of sphenoid sinus meningitis was considered to be a case of glaucoma and as such was referred to the Eye Service. The meningitis was then diagnosed and its origin in the sphenoid sinus was suspected, but the examining rhinologist did not find any nasal lesion. No gross bone changes were found on post-mortem examination and, unfortunately, the sphenoid bone was not studied microscopically. The pathway of infection from sphenoid sinus to meninges is therefore problematical, but bearing in mind the clinical and gross pathological findings, one may ascribe it to an infection along the path of the perforating vessels.

Case 2: F. O., male, age 50 years, admitted March 2, 1924, in delirium. He had had a "cold in the chest" several weeks before. Three days preceding hospitalization he became bed-ridden and was feverish. Two days later he was restless, delirious and later unconscious.

Physical Examination: Acutely ill, febrile, tossing about, comatose. Rapid, irregular respirations, moist rales at basis, Kernig, Brudzinski, bilateral Babinski, absent abdominal reflexes, rigid neck. Bilateral early papilledema, vertical nystagmus, enlarged heart, peripheral atherosclerosis. Provisional diagnosis, bronchopneumonia, bacteremia, pneumococcus meningitis, hypertension, atherosclerosis, emphysema.

Lumbar puncture yielded cloudy, green turbid fluid under increased pressure, 4,000 cells, all polymorphonuclear leukocytes, Gram positive diplococci with capsules. Bacteria were intra and extra cellular. Pneumococcus Type I. Temperature 103° to 104.6°. Respiration 72, Pulse 120, blood pressure 250/130. Type I pneumococcus serum

given intraspinally and intravenously. Patient died two hours after admission.

Post-mortem: Diffuse meningitis more marked at base, right cavernous sinus phlebitis, pus in both posterior ethmoid and sphenoid sinuses. Body of sphenoid hemorrhagic and purulent, red glazed pharynx and nasopharynx covered with dried purulent crusts. Pneumococcus Type I isolated in above-mentioned lesions,

This represents one of the fulminating cases of meningitis. The meningeal symptoms appeared 24 hours before death. No special examinations were made because of the death of the patient within two hours after admission to the hospital. The post-mortem findings of suppurative sinusitis and osteomyelitis of the sphenoid bone as forerunners of the intracranial lesions lead one to believe that they were of some duration, but they gave no symptoms at any time. In this case the pathway of infection was by direct continuity, from sphenoid sinus to spehnoid bone, cavernous sinus and meninges.

Case 3: A. S., female, age 55 years, admitted March 16, 1924, semistuporous. Four months before admission she fainted and had been bed-ridden ever since. She complained of headache, pains in the arms and legs, progressive weakness. The patient vomited frequently during the two weeks preceding admission to the hospital. She had lost 50 pounds in weight.

Physical Examination: Poorly developed, emaciated, lethargic, semistuporous, at times irritable, responds after repeated questioning. Mass in abdomen, stiff neck, Kernig on right side, Brudzinski, atherosclerosis, spondylitis, arthritis of fingers, hyperglycemia, R.B.C. in urine, spinal fluids 4,000 cells, 65 per cent polys., no bacteria.

Provisional diagnosis, metastatic malignancy from abdomen to brain and spine (possibly hypernephroma), diabetes.

Nasal Examination: Roomy nostrils, atrophy both inferior turbinates, hypertrophy of both middle turbinates, polypoid degeneration both middle meati, especially the left, pus both middle and superior meati. Beefy pharynx, marked secretion in nasopharynx. Diagnosis, chronic suppurative sphenoethmoiditis. Advised operation if no other source for meningitis was found. Ears negative. Fundi, myopia, hazy vitreous. W.B.C. 26,000, polys. 86 percent, hgb. 112 per cent, R.B.C. 6,100,000. March 18: X-ray showed all sinuses cloudy. Culture from sinuses and spinal fluid show organism of Bacillus Friedlander (ozena) group. Spinal fluid slightly turbid, under increased pressure, 1,550 cells, 70 per cent plys. March 19: Bilateral Kernig, retracted neck, right doubtful Babinski. March 20: All reflexes gone, spastic on left side, abdominal mass gone (probably

feces), spinal fluid 2,400 cells, 89 per cent polys. March 21: Right sphenoethmoidectomy showed thickened bone, small cells, muco pus.

Sphenoid small, mucosa swollen, pus present.

March 25: Left sphenoethmoidectomy showed polypi in middle meatus, pus in the posterior ethmoid, mucosa greenish-black, friable. Sphenoid contains blackish pus, mucosa not gangrenous, mucopus in anterior ethmoid region. W.B.C. 12,600, polys. 86 per cent. March 26: Spinal fluid 375 cells, 48 per cent polys. March 30: Left facial, spinal fluid 175 cells, 55 per cent polys. W.B.C., 10,600, polys. 72 per cent. March 31st: Signs of meningeal irritation of left hemisphere. April 3: Spinal fluid turbid, slightly increased pressure, 1,080 cells, 66 per cent polys. April 6: Intravenous administration of acriflavine 20 c.c. 2 per cent solution, followed by chill, later coma and death. The temperature ranged, as a rule, from 99.4° to 100.6°, going to 101° or 102° rarely. Pulse 112 to 120, respiration 24. Urine, very faint trace of albumin, sugar 0.2 to 0.8, occasional R.B.C. Blood sugar 0.170, blood culture negative.

Post-mortem: Purulent meningitis and encephalitis, large left sphenoid containing creamy, yellow pus, right sphenoid contained small amount of pus, as did the ethmoids on both sides. Posterior to the left sphenoid the medulla of the basisphenoid and basiocciput was soft and friable, with a path of necrotic bone leading from the left sphenoid sinus through the basiocciput perforating into the cerebellar fossa. The dura here was perforated. The surrounding dura was thickened, dull in appearance, covered with pus. The pia was adherent over the perforated dura. Culture: atypical Bacillus Friedlander group.

This case gave a history of intracranial disease of five months' duration. A diagnosis of metastatic malignancy was made at first because of coexistent lesions unrelated to the principal disease process. The examining rhinologist diagnosed sphenoethmoiditis and a bilateral operation was performed. The organisms found in the spinal fluid were the same as those in the sinuses, a group relative of the Bacillus Friedlander or so-called ozena group. After the intranasal operations the patient was markedly relieved for two weeks and the cells in the spinal fluid dropped from 2,400 to 175 and from 89 per cent to 50 per cent polys. The pathway of infection was from the larger sphenoid sinus, the left, through the medulla of the basosphenoid and basiocciput, through the dura of the cerebellar fossa and into the pia arachnoid. There had been, temporarily, a successful attempt on the part of the meninges to wall off the process in the region of the necrotic cerebellar floor, but this barrier was

finally broken down. The sinus origin of the meningitis once established, operative therapy was instituted, but as the post-mortem examination revealed, the intervention was not extensive enough to eradicate the disease. There was no indication of diseased bone in back of the mucosa of the sphenoids on operation.

Case 4: A. R., female, age 13 years, admitted Feb. 26, 1928, complaining of frontal headache of eight weeks' duration and stiff neck for seven weeks. Two months before admission to the hospital she had a cold in the head with severe frontal headache, nausea, vomiting, weakness, pallor and impaired vision. One week later chilliness and temperature of 105°. Temperature then came down to 102-103° for 10 days. The nose was obstructed at first and frontal tenderness was present. Pyuria.was found. Six weeks before admission there was marked hematuria. I saw the patient then, in consultation with her physician who was looking for a focus for the hemorrhagic nephritis, and found a bilateral ethmosphenoiditis. One week later pain in the back of the neck and drowsiness appeared, and two weeks later spinal tap revealed increased pressure and 20 cells. Loss of vision and drowsiness increased. One week before admission, diagnosis of an inflammatory lesion at the base of the skull was made.

Physical Examination: Semistuporous, but could be aroused; wasting and loss of power in muscles. Amaurotic, papilledema with hemorrhages and dilated veins. Dilated pupils, left greater than right, fixed to light. Deep reflexes and abdominals absent. General examination negative; mild meningeal signs, left external rectus weak, superior recti and right inferior rectus paretic, slight retraction of neck, slight weakness of right face, nasal speech. One neurologist diagnosed basilar meningitis of sphenoid origin, another diagnosed toxic polyneuritis. Feb. 24: Slight double Kernig with rigid neck. Urine, occasional W.B.C., no R.B.C. Feb. 29: Nose, crusts and secretion both sides. X-ray of sinuses negative. W.B.C. 10,600, polys. 85 per cent. Spinal tap showed fluid under low pressure, 20 cells, 90 per cent lymphocytes. Temperature was normal till day before death. Pulse 90-120. Seven days after admission temperature went to 104°, pulse 180. Patient became comatose and incontinent and died.

The post-mortem findings, which will be demonstrated by Dr. Druss, were purulent sphenoidal sinusitis, basilar meningitis, especially in the interpeduncular spaces and about the chiasm, tremendous right frontal lobe abscess. The pathway of infection was through the roof of a pneumatic process of the right sphenoid. From the

roof of this extension the infection passed through the dura and involved the right frontal lobe.

This case is unusual, inasmuch as it presents a brain abscess of sphenoid sinus origin. There is only one other in the literature. Before admission, the patient was thought to have a hemorrhagic nephritis, at which time I saw her because a focus of infection was being sought. The patient presented signs of a mild sphenoethmoiditis, for which conservative treatment was advised. No other nasal examination was made until shortly before death. The patient had been in two hospitals, where a variety of diagnoses had been made. The basic lesions, sphenoidal sinusitis and basilar meningitis had been recognized, but not the frontal lobe abscess. Despite its enormous size, no focal symptoms of the abscess were evident.

Case 5: L. M., female, age 53 years, admitted Feb. 21, 1928, with a story of headache for one month and coma for three days. For the past four or five months she had vomited intermittently; one month ago she had had severe pain in the back of the head; two weeks later she became weaker and had a septic temperature up to 104.2°. Three days before admission she became unconscious, but awakening at times she was then found to be blind. Fever and nasal discharge were present. Nine years before, she had had an operation on the right eye and became blind in the right eye six years later. Fifteen years ago the right ear was operated upon.

Physical Examination: Acutely ill, obese women, unconscious. Right eye covered with gray exudate, obscured gray cornea, dilated conjunctival vessels; left eye less involved. Nose was filled with secretion; right ear full of pus; dry glazed throat, exudate on posterior pharyngeal wall; rigid neck, absent abdominal reflexes, exaggerated deep reflexes, bilateral Kernig and Babinski; spinal tap yielded slightly cloudy, faintly yellowish fluid, normal pressure, 27 cells, mainly lymphocytes, temperature 101°, pulse 120, respiration 20, blood culture negative, spinal fluid culture, streptococcus gamma (a slow growing organism). Provisional diagnosis: Pansinusitis and ophthalmitis, brain abscess, secondary meningitis. Patient died in 12 hours.

The post-mortem findings will be presented by Dr. Druss. They were, briefly summarized, the following: Pus in left antrum and some of the ethmoid cells, sphenoid sinus absent. In the body of the sphenoid there was a sinus tract which ran from the roof of the nasopharynx through the sphenoid bone in an upward and backward direction for a distance of 1.5 c.m. It was 0.5 c.m. in diameter at its widest portion and was filled with detritus. The sella turcica was

eroded and the periosteum over the sphenoid bone at the base of the skull was considerably thickened and in places it was lifted from the underlying bone. Basilar meningitis, hypophysitis, ependymitis, purulent bronchopneumonia, old T.B.C.

This unusual case showed an infection of the sphenoid bone originating in the craniopharyngeal sinus or the anlage of the anterior lobe of the pituitary. The infection extended through the sphenoid bone, eroded the base of the skull and produced a basilar meningitis. The patient's symptoms dated back four or five months when she began to vomit. Whether this was the first symptoms of meningitis or of sphenoid bone involvement, is problematical. The subsequent symptoms, pain in the back of the neck and septic temperature can also be referred to either the meningitis or the osteomyelitis. The ante-mortem diagnosis of osteomyelitis due to Rathke's pouch infections seems to me to be an impossibility. Perhaps some inkling of the presence of the osteomyelitis might have been obtained by palpating the inferior surface of the sphenoid through the nasopharynx and in this way eliciting tenderness or swelling. The patient died so shortly after admission to the hospital that extended study was impossible. Unquestionably, the ear infection would have been a source of confusion in the diagnosis and therapy of this case, as has happened in many other sphenoid bone infections. One phase of the history might be significant in the clinical diagnosis, and that is the persistent pain and stiffness in the back of the neck, which should arouse suspicion of sphenoid bone infection.

Case 6: F. W., male, age 52 years, admitted Dec. 16, 1928, in coma of one day's duration. One week before admission he had had an upper respiratory infection, which became worse five days later. He then had generalized headache, restlessness, impaired hearing; one day later had generalized convulsions followed by coma. Following scarlet fever and meningitis he had become blind in childhood.

Physical Examination: Febrile, comatose, thrashing about, mumbling incoherently, optic nerve atrophy. Eardrums full and red, landmarks visible. Left facial paralysis, stiff neck, retracted head, absent abdominals, knee and ankle jerks, Kernig and Brudzinski present, no Babinski.

Provisional Diagnosis: Otitic meningitis. Blood pressure 140/80, temperature 102° to 106.4°. Spinal tap shows turbid fluid 5,000 cells, 100 per cent polys., Gram positive, encapsulated cocci, pneumococcus Type III. Blood culture, positive Type III pneumococcus. Otlogist found mild bilateral otitis media and suggested otitic or nasal

origin of meningitis. No nasal examination was done because of death of patient shortly after admission.

Post-mortem: Purulent meningitis, empyema of very large sphenoid, parenchymatous degeneration of heart, liver and kidneys, thrombosis of left external iliac vein, pulmonary embolus and multiple infarcts, arteriosclerotic kidneys, acute splenic tumor.

This was a fulminating meningitis following sphenoid sinus suppuration, the first symptoms of meningeal irritation appearing two days before death. The upper respiratory infection, which was the apparent cause of the sphenoid involvement, started five days before. The pathway of infection from sphenoid sinus to meninges was not evident macroscopically. Dr. Druss is studying the microscopic sections and will report his findings at a later date. One may, at present, hazard the assumption that the meninges were infected by way of the perforating vessels in the sphenoid sinus walls. This case is another example of blood stream infection associated with meningitis of sphenoid origin.

The following three cases had no cranial post-mortem examination. Case 7: S. B., male, age 15 years, admitted May 4, 1928, complaining of pain in the back of the neck for four days. Four weeks before, patient had headaches and loss of appetite and malaise; 10 days later his temperature was 105°. Four days before admission pain in the back of the neck appeared, associated with stiffness and limitation of motion; no discharge from nose but nasal obstruction and mouth breathing present.

Physical Examination: Stiff neck, limited motion, tenderness of dorsal muscles and cervical spine. Systolic murmur over heart, enlarged spleen, secretion and congestion of nasal mucosa, secretion in Diagnosis, sinusitis, sepsis, cervical spondylitis. nasopharynx. W.B.C. 7,000, polys. 70 per cent, hgb. 62 per cent, temperature 100°. May 7: Temperature 103° to 100°. Pain on right side of head, congestion right drum, tender over antra and ethmoids. Blood culture Gram positive cocci in chains. Ear examination by otologist, right ear negative except for slight congestion and fullness around Shrapnel's. Nose examintion: Septum markedly deviated to the right, pus right middle meatus and in nasopharynx. Fundi negative. May 9: W.B.C. 16,000, polys, 61 per cent. Because of signs of sepsis and meningeal irritation, operation on the sinuses was decided upon. A partial submucous resection and bilateral sphenoethmoidectomy was done under local anesthesia. The submucous resection was necessary because of marked right nasal obstruction, making the approach to the right ethmoid and sphenoid impossible. Inspissated pus was found in the right anterior ethmoid, from which staphylococcus albus was cultured. There was a large amount of thick pus in the left sphenoid, from which streptococcus hemolyticus was obtained. May 10: Somewhat better except for pain and acute tenderness in right costal margin near xyphoid. May 11: More toxic, temperature 103.4°, meningeal signs more marked. May 12: Worse. May 14: Exitus. Spinal tap 450 cells, 80 per cent polys., streptococcus hemolyticus. X-ray of sinuses negative. Urine showed traces of albumin, W.B.C. and granular casts. Post-mortem showed streptococcus hemolyticus spinal meningitis, parenchymatous degeneration of heart, liver and kidneys. No cranial post-mortem.

This boy presented the picture of sepsis and meningitis; the left sphenoid sinus showed pus due to the same organisms as those found in the blood and meninges, the streptococcus hemolyticus. We have here another example of meningitis and sepsis associated with a sinusitis. Whether they both arise at the same time from the sinusitis by invasion of the vessels in the bony walls, or one is dependent on the other cannot be determined definitely, but their simultaneous occurrence would seem to indicate their common origin. Drainage of the infected sinus was of no avail. In the absence of a cranial post-mortem examination the pathway of infection is one of speculation.

Case 8: R. G., female, age 16 years, admitted Oct. 31, 1924, with headache for two days and vomiting, coma and convulsions for one day. Two weeks before she had been ill, but had attended school. She lost weight and had poor appetite. Twelve days later she had frontal headache, vomited and could not sleep. The next day she was mentally confused, tossed about, had slight convulsions, later coma, and then recovered somewhat. Temperature was 102.6°. She had a history of rheumatism and heart involvement.

Physical Examination: Acutely ill, slight ptosis of eyelids, injected throat, systolic murmur at apex, rigid neck, hyperactive reflexes, positive Kernig, right Babinski, tender right frontal. Provisional diagnosis: Meningitis. Spinal tap, turbid fluid, increased pressure, 4,290 cells, 94 per cent polys., culture pneumococcus. Ear examination negative. Nose examination, by two rhinologists, negative; a third found bilateral ethmosphenoiditis and advised operation. W.B.C. 23,400, polys. 89 per cent.

Nov. 2: Spinal tap, 4,560 cells, 98 per cent polys. Nov. 3: Spinal tap, 640 cells, 90 per cent polys. Nov. 4: Spinal tap, 420 cells, 90 per cent polys. Nov. 6: Vomited, neck rigidity less. Nov. 7: Spinal tap, 99 cells, 87 per cent polys. Nov. 8: Better, temperature

lower, W.B.C. 12,200, polys. 73 per cent. Nov. 10: Neck rigidity less marked, Kernig and Babinski present. Nov. 17: Frontal headaches past two days: signs still the same, increased frontal tenderness. W.B.C. 28,400, polys. 96 per cent. Spinal tap, increased pressure, turbid, 7,540 cells, 96 per cent polys. Neurologist states that signs point to a meningoencephalitis, and not to a brain abscess. Left fundus is blurred, vessels injected, right fundus negative. Beginning left optic neuritis. Nov. 18: Re-exploration of right paranasal sinuses, frontonasal duct free, no secretion in frontal sinus, culture from ethmoid region Gram negative bacillus and staphylococcus aureus. Nov. 19: Signs same, mental condition good, frontal tenderness less marked, spinal fluid, 5,880 cells, 98 per cent polys., culture Gram positive diplococcus. Nov. 20: Temperature elevated, pain in hamstring muscles, spasmodic severe headache and frontal tenderness marked, large doses of morphin required for pain. Nov. 21: Because of lack of relationship between meningeal signs and right-sided sinuses, it was decided to explore sinuses on left side. Left ethmosphenoidectomy, local anesthesia, thick pus in sphenoid, otherwise negative. Culture pneumococcus, type not determined. Nov. 22: Spinal fluid, 3,160 cells, 98 per cent polys., Gram positive diplococcus (pneumococcus). Intravenous administration of Felton's pneumococcus antibody. Ears negative. Patient worse, vomited and was irrational. Increased left optic neuritis. Administration of Felton's serum. Nov. 24: Vertigo nystagmus, ears negative harsh systolic at apex. Nov. 26: More marked left optic neuritis, more irrational. Nov. 27: Exitus. No post-mortem examination was obtained. Temperature ranged from 100.2° to 104°, never normal. Pulse 80 to 100, respiration 20.

The intracranial conditions must remain a matter for speculation, inasmuch as no autopsy was performed. This patient had a bacterial meningitis for one month, most probably limited to the base for the greater part of this time. Whether her frontal pain and tenderness was an expression of a frontal lobe abscess, as in Case 4 or an osteomyelitis of the sphenoid in a matter for discussion. There was no evidence of frontal sinusitis. The course and the operative findings indicate the presence of a left sphenoidal sinusitis with subsequent osteomyelitis of the sphenoid bone and basilar meningitis. The right ethmoid was diseased but the organisms found were not the same as those present in the spinal fluid, whereas those found in the left sphenoid were the same; that is, a pneumococcus. The involvement of the left fundus with a normal right fundus also points to a lesion in the left sphenoid. Clinically one must conclude that the left sphe-

noid sinus was the origin of the intracranial process. In this case, we have another example of the failure of the rhinologists to diagnose the sinus disease when present in a case of intracranial infection. The operative therapy, the drainage of the involved nasal sinuses, was totally ineffective in checking the progress of the disease. It is true the causative lesion was treated late in the course, but it is doubtful if greater success would have crowned a similar procedure if employed immediately after the diagnosis was made.

Case 9: B., male, age 11 years, admitted March 2, 1928, with a history of an upper respiratory infection three weeks before with discharge of pus from the right ear, and high temperature, which subsided until five days before admission. He then had a chill and high temperature, vomiting, headache, rigid neck. For five years the child had had a right otitis on severa loccasions. Provisional diagnosis of mastoiditis or sinus thrombosis was made. Blood culture was negative. For a few days his condition improved, and then a hemorrhagic glomerular nephritis developed. This subsided; then the temperature rose again, neck became rigid, and nasal and postnasal discharge appeared.

March 13: W.B.C. 9,000, polys. 57 per cent, hgb. 90 per cent. March 13: W.B.C. 28,600, polys. 75 per cent, hgb. 75 per cent; urine, albumin 4+, waxy and other casts, R.B.C. March 13: Nose, purulent secretion both sides in all meati, congestion of mucosa, hypetrophy both middle turbinates. Pharynx congested, secretion posterior pharyngeal wall. Diagnosis: Bilateral ethmosphenoiditis.

X-ray of sinuses, right antrum, ethmoid and sphenoid cloudy. Otologists stated that the ear did not cause condition. Decided to wait another 24 hours and use conservative sinus treatment. March 14: Nose: pus still present in right superior and middle meati. Edema of right eyelids and cheek. Fundi, veins engorged. W.B.C. 21,400, polys. 60 per cent. Advised intranasal exploration of ethmoid and sphenoid. Operation refused. March 15: No clinical signs of mastoiditis. Slight discharge from ear. Otologist insists that ear bore no relation to meningeal condition. Nose same as on March 14. Another rhinologist in consultation stated that there was no evidence of sinus disease and was absolutely certain that meningitis could not arise from sinus, but probably came from right ear infection. Right mastoid region explored and found normal. Spinal tap, 500 cells, 95 per cent lymphs., culture streptococcus hemolyticus. March 16: Developed severe meningeal symptoms, cisternal tap, 1,000 cells. March 21: Cortical irritation and meningeal process at base. Temperature 104°; exitus. No post-mortem examination was

permitted, but an intranasal exploration of the sinuses was performed. On opening the right sphenoid, pus under pressure escaped. There was polypoid degeneration of the right ethmoid and pus in the right antrum.

This patient had signs of meningeal irritation for three weeks. with organisms in the spinal fluid one week before death, when the first spinal tap was done. The complicating hemorrhagic nephritis was probably due to the infecting organisms, the streptococcus hemolyticus, as is quite often found in bone infections by this coccus. The focus from which the meningitis took its origin was in dispute, because, here again, as in other cases, an otitis and a sinusitis were present. The otologists who had had a great deal of experience with intracranial complications of otitic origin were emphatic in their statements excluding an otitic origin in this case. The sphenoid sinus appeared to be the definite cause, but, as in other cases recited, a rhinologist in consultation denied the existence of a sinusitis in this case and the mastoid region was explored, with negative findings. Still the consulting rhinologist insisted on the impossibility of a nasal sinus involvement as a focus. Exploration of the nasal sinuses showed a purulent right sphenoidal sinusitis.

The usual pathways of infections from the sphenoid sinus or bone to the intracranial contents are as follows: *I*. By way of the blood vessels perforating the bony wall. *2*. By way of the general blood stream. *3*. Through dehiscences in the bone. *4*. Through necrosis of the bony walls. *5*. Through osteomyelitis of the sphenoid bone. *6*. Through involvement of the orbit, and then through the orbital veins, the orbital foramina or fissures, or through the orbital roof.

In Case 2 the infection was by way of an osteomyelitis of the sphenoid with subsequent cavernous sinus phlebitis and meningitis. Case 3 showed an osteomyelitis of the sphenoid with involvement of the cerebellar fossa. Case 5 showed an osteomyelitis of the sphenoid (no sphenoid sinus present but a persistent craniopharyngeal canal) with involvement of the hypophsis and adjacent territory. Case 4 showed a necrosis of the roof of a pneumatic process of the sphenoid. Case 1 was subjected to no microscopical examination, but the gross appearance showed no definite path of infection. This is the usual finding in lesions transmitted by the blood vessels. Case 6 is one similar to Case 1. This is being studied microscopically at present.

The following organisms were found: Cases 1, 7 and 9, strepto coccus hemolyticus. Case 2, pneumococcus Type I. Case 3, bacillus of Friedlander group. Case 5, streptococcus gamma. Case 6, pneumococcus Type III. Case 8, pneumococcus, type not determined.

The affected sphenoid sinuses were all large. The following intracranial complications were found: meningitis, in every case, most frequently limited to the base; frontal lobe abscess; right cavernous sinus phlebitis; encephalitis and ependymitis. The proportion of cavernous sinus phlebitis to meningitis in this small series is much lower, one to eight, than in the series of cases collected by Burger, where they are found to be of approximately equal frequency.

Grouped according to duration, these cases fall into three classes: the fulminating, the acute and the chronic. The fulminating cases gave a history of 24 to 48 hours of illness before death; the acute cases lasted from a few days to two or three weeks, and the chronic cases were several months in duration.

The fulminating type presented symptoms of a violent meningitis coming on suddenly after a few days of a preceding upper respiratory infection. They were usually comatose or delirious in a short time and the spinal tap gave positive evidence of a bacterial meningitis. Papilledema was present in one case, ophthalmitis in another. Death ensued a few hours after admission to the hospital. Usually the diagnosis lay between an otitic, a pneumococcic and at times an accessory nasal sinus meningitis. Active intervention was not undertaken in any of these cases.

In the cases of longer duration, several features were outstanding. The first was the presence of headache, at first frontal but later occipital. This was present in all the acute and chronic cases. Vomiting was a symptom in almost all these cases. Eye changes, such as papilledema, optic neuritis, homonymous hemianopsia, amaurosis, appeared in five of seven cases. Meningitic signs were present in all. These were variable in the chronic cases and at times might be very few and slight. Rigid neck and positive Kernig were present in all cases, but these varied in degree from day to day. The other meningitic signs varied in their appearance and degree. The spinal tap gave positive evidence of meningitis in all cases except in two, in which a low count more in keeping with a brain abscess than with meningitis, was obtained. The culture of the spinal fluid was positive in all cases except one in which a cell count of 30 was found and in which a basilar meningitis and brain abscess were found on postmortem. The examination of the nose revealed definite findings of an ethmosphenoiditis in six cases out of seven studied. In three of these six positive cases, four different rhinologists had failed to make the rhinological diagnosis. X-ray examinations of the sinuses were negative in all except two cases. Blood culture was positive in three cases. In four cases the ears showed infection which had to be considered in determining the origin of the intracranial lesions; and in one case, mastoid bone exploration was done, with negative findings.

In the diagnosis of these cases one must bear in mind that very few symptoms may be present and that nasal and X-ray examination may be negative. The diagnosis can only be made then by exploration, with the exclusion of other foci of infection. In the case of sphenoid osteomyelitis from Rathke's pouch infection, digital examination of the vault of the nasopharvnx and exploration of the sphenoid bone are the only methods that might aid in the diagnosis. The presence of definite sinus involvement in cases of intracranial infection calls for serious consideration of the infected sinus as a focus. From a study of cases such as those described in this paper, one gets the impression that osteomyelitis of the sphenoid is accompanied by headache, either frontal or occipital, or both, by some stiffness of the neck and by occasional vomiting. This has not been definitely verified; nor do they appear only in osteomyelitis, inasmuch as an occasional uncomplicated sphenoid sinusitis or a basilar meningitis present these symptoms, but it seems to me to be a good working basis for the establishing of the diagnosis and for the therapy of osteomyelitis of the sphenoid before serious, perhaps irremediable intracranial complications have arisen. The persistence of mild meningitic signs in addition to the above (sinusitis, headache, vomiting, stiffness in neck), and the finding in the spinal fluid of indications of infection are definite evidences of an intracranial complication of probable sphenoid origin. The ears often are a cause of confusion and at times one is forced to explore the mastoid in order to exclude or establish the existence of an otitic focus. This is a very frequent occurrence in cases of sphenoid origin, as evidenced by Burger, who in 13 collected cases of meningitis of sphenoid origin found that six had had otological operations and the sphenoid origin was not discovered until the post-mortem had been performed. Pneumococcic meningitis of pneumonic origin may be a source of confusion and the meningitis may be ascribed to a pneumonia instead of a sphenoid infection as in Case 2. The differential diagnosis of the type of intracranial lesion, meningitis, abscess or sinus phlebitis, is too broad a subject to be taken up in this paper but I want to emphasize the lesson one can learn from the cases reported here, and that is that meningitis may be of long standing, that the symptoms may be vague and easily overlooked, that they may simulate a brain abscess by their mildness and chronicity, and that brain abscess and cavernous sinus thrombosis may be entirely symptomless in the presence of other lesions or simulate other intracranial lesions.

The prognosis is bad because of the difficulties in the way of satisfactory surgical approach.

The prophylaxis offers material for serious consideration. Many cases present no history of previous sinus involvement and the intracranial lesions appeared, as far as can be determined, a very short time after the initial involvement of the sphenoid. In these cases, prophylaxis seems an impossibility. On the other hand, a number of cases gave a history of long standing sinusitis. Should one, therefore, adopt the policy of opening widely all infected sphenoids that persists for more than a short period of time? Would intracranial complications be avoided in this way, and would the fatalities resulting from widespread operation equal that of the complications of neglected sphenoid infections? One point must be borne in mind, and that is the size of the great majority of offending sphenoid sinuses has been large and drainage through the natural passages has been unsatisfactory.

The therapy of these conditions has been far from gratifying. The surgical approach by the external route, whether it be temporal, orbital or frontal, is exceedingly difficult and carries with it enormous dangers, both from the standpoint of trauma and spread of infection. The intranasal and intraoral routes do not permit of proper inspection of the intracranial lesions and they afford access of infection to the intradural structures. The main attempts from the operative standpoint have been to open the sphenoid sinus widely and treat the intracranial lesion symptomatically. There are, in the literature, three cases of intracranial lesions due to sphenoid infections that have recovered after drainage of the sphenoid sinus. It seems to me that in cases where a bone lesion of the sphenoid is suspected, that a feasible procedure would be to open up the sphenoid sinus, remove the mucosa and explore the bone for areas of infection. The osteomyelitic portions could be curetted out to their limits. This would serve in removing the focus from which the intracranial lesions have arisen in these cases. The intracranial lesions may be attacked in the manner that one is accustomed to treat such lesions; that is, a brain abscess if diagnosed and localized as frontal may be approached transfrontally or temporally; a cavernous sinus approached by the Kronlein or Eggleton route. But the primary focus must be eradicated if any results are to be expected. The results from the use of vaccines, sera and chemicals have also been discouraging in spite of recoveries ascribed to their use in the treatment of intracranial infections. Our best efforts should be directed towards the early diagnosis in the less acute cases, so that drainage of the sphenoid

and removal of osteomyelitic foci may offer greater prospects of a cure.

The solution of the various problems associated with these conditions depends on a close co-operation between the several specialties. The internists and neurologists must bear in mind the possibility of sphenoid infection in cases of meningitis, the rhinologist must make the diagnosis of sinusitis and either he or the neurological surgeon, or both, must devise a method of appropriate and satisfactory treatment. The department of pathology will have to co-operate in obtaining the post-mortem examinations of all meningitis cases and in determining the proportion of these cases to uncomplicated unoperated sphenoid infections. The rhinologist will have to decide for himself at present whether he shall interfere in all cases of sphenoid sinusitis so as to prevent intracranial involvement at a later time.

121 East 60th Street.

UPPER RESPIRATORY THERAPY IN PERSISTENT HICCOUGH.*

DR. V. K. HART AND DR. J. P. MATHESON, Charlotte, N. C.

The medical profession has recently been scathingly criticized by a layman. The censure also came from a lay magazine, the *Cosmopolitan*. Nevertheless, there was some justice in the plea for attention by the profession to relief of annoying, and sometimes distressing discomforts, regardless of diagnosis. The fact that the plea is from a lay person takes on connotation.

Hence the following is presented in the hope that it may be of use to colleagues in relieving a symptom-complex more or less common, but of great mental and physical suffering to the patient. This simple procedure is not infallible, but often gives prompt and lasting relief, and an ever-grateful patient.

It is applicable to any patient with persistent hiccough. Success is probably most conspicuous in those patients with some infection of the larynx and trachea. However, curiously enough, it has been very effective without obvious infection.

The technique is simple. The throat is sprayed with a 2 per cent cocain solution. One application of 20 per cent cocain is then

^{*}From the Charlotte Eye, Ear, Nose and Throat Hospital.

made to the larynx by indirect laryngoscopy. Again by indirect laryngoscopy, about 1 c.c. of 4 per cent cocain in a laryngeal syringe is instilled drop by drop between the cords into the trachea. By the same method, warm oil (plain albolene), is then slowly dropped into the larynx and trachea.

Two cases are briefly cited to illustrate the use and effect of the above treatment.

Case 1: Physician in early seventies. First seen March 20, 1929, because of cough and persistent hiccough of short duration. History of recent upper respiratory infection. Physical examination had been essentially negative. Laboratory findings showed nothing of value. Ear, nose and throat examination disclosed only a probable tracheitis. Treatment always promptly relieved the patient for as long as 12 hours. He was treated once daily for five successive days and entirely relieved of both cough and hiccough.

Case 2: Male, age 41 years. First seen April 25, 1929. Had had hiccough for 10 days with only a few short periods of relief. Past history negative and no history of recent cold. Patient very nervous and more or less exhausted. Here, too, all laboratory work (both urine and blood, and of course a Wassermann), had been negative. A complete gastrointestinal X-ray had also been negative. The physical examination was negative. Ears, nose and throat examination showed a deflected septum and a large, diseased pair of tonsils. The larynx and trachea showed no obvious pathology. Still, the patient was at once relieved for two hours by the first treatment. The same result followed a second treatment the same day. Treatment once daily for two days thereafter, completely and permanently relieved the patient. He has remained well since.

Comment: Some six or seven cases have been observed in this clinic and all relieved by the above treatment. Practically all showed some respiratory infection except Case 2, as heretofore cited.

When infection is present in the throat, larynx, or trachea (particularly the trachea), cocain abolishes the afferent source of a reflex irritation via the pneumograstric. The efferent are of course through the phrenic from the third and fourth cervicals.

In the absence of infection, the rationale of the treatment cannot be explained, except from some obscure respiratory irritation or through some action of the sympathetic, a few fibres of which supply the trachea with the tenth. This latter suggestion is far-fetched, with no scientific basis.

There is nothing unusual about the treatment. Its very simplicity bespeaks its trial. After all, we are prone to forget such therapy in our search for a diagnosis or an operative procedure.

THE WINDOW OPERATION FOR HEMATOMA AURIS AND PERICHONDRITIS, WITH EFFUSION.*

Dr. Robert C. Howard, New York.

Acute fluid swellings beneath the perichondrium of the anterior surface of the auricle are often refractory to treatment and unsatisfactory in results. They are produced most frequently by a blow on the ear, and are much more common among boxers than in any other class; although found in football players, the insane and aged. Also in piano movers from pressure on the ear in lifting, as was the case with a patient in my series. The usual modes of treatment are often tedious, prolonged and unsatisfactory; both to the patient and surgeon, requiring a great deal of time for dressings, and resulting in considerable disfigurement to the patient.

With these thoughts in mind, nearly two years ago I decided to apply to these auricular conditions an operative treatment which had proved most satisfactory in dealing with almost identical lesions in the nasal septum.

In the past 14 years I have used, in one way or another, the method of punching windows out of the muco-perichondrial layer of the septum, to afford prompt and efficient drainage of abscesses and hematomata of the nasal septum: either following the submucous resection; injuries to the nose resulting in these conditions; or others of unknown etiology in a great many instances, and I have found it the simplest as well as the best way of dealing with these troublesome complications.

The possibility of deformity of the nose from absorption of the cartilage of the septum, and the removal of this support from the bridge, I am sure, is much less where this method drainage is used than where simple incision and a drain of gutta percha, rubber dam or gauze is employed.

This rhinological background dealing with a great many patients with analogous conditions, treated almost identically and observed, many of them, for years especially as to deformity and results, has a definite bearing, I feel, on the present otological paper, which is very much in the nature of a preliminary report; in that it deals with comparatively few patients; with the aural condition in question and

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the study of the ultimate results of the operations are incomplete in certain respects and cover too short a period of time to be conclusive.

Description: Hematoma auris or othematoma, and perichondritis although usually described as separate conditions, they seem to have the same etiology—trauma being the causative factor in each. The fluid in perichondritis is more apt to be transparent and straw-colored; although usually with evidence of blood intermixture; but varying with the stage and character of the process. On transillumination the light passes through more freely than in the case of hematoma, where clouding is much more pronounced. In both conditions there are evidences usually of chondritis, perichondritis and extravasation of blood and serum between the cartilage and perichondrium.

The tumefaction is most often on the outer surface of the auricle, where the integument and perichondrium are more intimately fused than on the inner or posterior surface, where they are more loosely connected, and the effusion when it does take place there is more apt to be in the loose areolar tissue, where it is more readily absorbed, with less tendency to organization, thickening and deformity.

The cavum conchae and triangular fossa are perhaps more frequently the site of the localized swelling; although the whole outer surface, except the lobule, may be involved and even the inner surface of the pinna, in certain instances.

When infected, the process is much more severe, and the signs of inflammation—redness, heat, pain and swelling—are very pronounced and call for prompt and radical operative measures.

The swelling is usually more or less globular, and of a bluish-red color, definitely fluctuating to touch except in the early stages, where the clot is still firm, and later when organization of the clot and permanent thickening of the perichondrium and adjoining structures have taken place. In most instances, if seen a week or more after the injury has occurred, the cartilage will show signs of necrosis and absorption, as evidenced by loss of luster, roughening of surface, greater flexibility and loss of resiliency. This may be very slight or marked and varies with the stage and severity of the process. Absorption taking place much more rapidly when the fluid is purulent and the pain severe. When not infected, the pain, tenderness and fever are slight or of moderate degree. Purulent perichondritis may result from furunculosis of the auricle or follow the radical mastoid operation. Trauma is nearly always the cause and a blow struck while boxing is by far the most common cause; although degeneration of the blood vessels, as in the aged and insane; and certainly

hemophilia are predisposing factors. Pressure of the ear against a hard surface, as occurred in the case of one in my series, a piano mover, who was accustomed to rest the side of his head against the wood when lifting upright pianos.

Treatment: When seen immediately after the injury has occurred, firm pressure of the hand against the ear with compresses or a towel interposed, saturated with ice water, should be employed for 20 minutes or more to localize and limit the bleeding; also cold compresses of boric acid solution, lead and opium wash or solution of aluminum acetate should be kept applied, although frequently changed for the first 24 hours or more, after which warmth may be used.

In a few days, if the swelling is small or shows a tendency to definitely diminish, expectancy may be employed; however, usually after three or four days, unless diminution is definite and rapid, or the exudation very limited, it should be freely opened.

The Window Operation: The skin of the auricle is sterilized by the application of half-strength iodin tincture and the excess removed with alcohol, very much as when preparing for the mastoid operation, although the field is more limited and the side of the head over the temporal, parietal and mastoid regions are not included. The patient is draped following the usual operating room technique. A sterile solution containing 1 per cent of procain and a trace of adrenalin chlorid (1:50,000) is injected beneath the epidermis of the area to be excised, and this is usually at the most dependent part of the fluctuating mass. So far as feasible the window should be placed so that the resulting scar will be hidden, as much as possible, in a fold or shadow. A short incision, at right angles to the long axis of the window to be removed, large enough to permit of entry of the punch, is now made through the skin and perichondrium. With a Gruenwald punch a section of tissue is bitten out of the skin and perichondrium en masse, and the fluid expressed. If there is evidence of thickening the cavity should be curetted with a Spratt's curette or other suitable instrument. Loose pieces from fracture or definitely sloughing cartilage should be removed or its surface scraped if indicated.

The size and number of the fenestrations will depend on the extent and character of the swelling. If obviously infected, showing a tendency to spread and of considerable size, a number of windows should be made and placed to give the best drainage with the lowest visibility of the scar kept in mind. The window will stay open about three weeks and this is usually sufficiently long to prevent backing up or refilling.

If the tissues are not inflamed and the fluid not infected, a cottoncollodion cast is very satisfactory; but if there is any sign of active inflammation the cast should not be used and instead sterile dressings in the usual way should be applied. However, if, as is often the case, the walls collapse on opening and there is little or no induration left after removing the fluid, a very convenient and satisfactory dressing consists of a collodion and cotton cast formed by painting a number of layers of collodion with thin strips of cotton applied to hold the collodion and then a thick layer of cotton placed behind the ear; also a firm ball of cotton is applied to keep the outer wall tight against the cartilage while the cast is hardening and a snug bandage is applied to cause the cast to harden with the proper confirmation to the parts and to give apposition of the tissues. Cotton must be used freely on top of the collodion, as gauze is difficult to deal with later. The excess cotton can easily be removed, but gauze gets entangled in the collodion and is hard to remove. The window must be left open and the collodion is only applied around it. A small pledget of sterile gauze is lightly placed over the fenestration. Subsequent dressings are simple. The following day the bandage is removed for inspection; the excess cotton separated and, if necessary, retouched with collodion; the sterile gauze is removed from the opening and if in good condition, as is usually the case, simply a 2 per cent solution of mercurochrome is applied to the wound and a small sterile dressing of gauze placed over the fenestra and held in position by short adhesive strips against the cast.

The ear is now partly encased in the collodion cast and is movable. Patient may rest or sleep upon it without fear of harm resulting and the subsequent visits for inspection and dressing are very simple and free from pain as a rule. It may only be necessary to apply the cast to the outer surface of the ear; but usually a better apposition is obtained if it is extended over the helix to the posterior surface, also better protection is thereby afforded. The cast is ordinarily kept on till the wound is practically healed, which varies from two to four weeks. Should pain or other untoward symptom develop, the cast is readily removed by softening with a mixture of three parts ether and one of alcohol. If there is any sign of infection or much induration, the cast should not be used in the first place.

The only contraindications I know of are lack of positive indications, such as when the swelling is small or where it tends promptly to regress under suitable conservative treatment.

I have employed this operation in five cases and my associates in two additional ones, making a total of seven, and the results of this window operation, so far as it has been possible to determine, have been very satisfactory and with very little scarring or deformity and with no untoward result.

CONCLUSION.

The method of removing a piece of tissue consisting of perichondrium and a full thickness of skin by means of a punch and thus producing a window with prompt evacuation of contents and free drainage appears, from present indications, to be a satisfactory way of dealing with these hematomata and fluid formations between the perichondrium and cartilage of the auricle.

839 West End Avenue.

DEMONSTRATIC : OF INTRATRACHEAL ANESTHESIA AND A NEW METHOD OF RESUSCITATION.*

DR. PALUEL J. FLAGG, New York.

Owing to the short time at my disposal, it will not be possible to bring out here more than the high points of the subject under discussion. With your permission, I will reverse the order on the program, and speak first of resuscitation, following with a few remarks on intratracheal anesthesia.

The subject of resuscitation has been more or less neglected by the practitioner. Perhaps this is due to the fact that the methods suggested today may be used by the layman, and most of us do not like to employ as a medical practice what any layman can use with equal advantage. However, it would seem that we can place the problem of resuscitation on the same basis and on the same high medical plane of excellence as is done in the examination of the patient in heart disease. We are all familiar with the three cardinal points in the examination of heart cases; namely, inspection, palpitation and auscultation. The same general plan should be applied to resuscitation, where we should carry out as a routine practice the three principles of inspection, aspiration and insufflation.

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Until recently we have not had a practical and reliable means of carrying out inspection, for we have been forced to depend for the inspection of the larynx on the delicate elements devised by Dr. Jackson and others. The tiny electrical bulbs blow out so frequently that we could not put ourselves in a position to depend on an instrument which was likely to fail at a critical time. Therefore, the proper apparatus for inspection is the turning point of the procedure. We have finally assembled what may be described as a pocket flashlight larvngoscope. This larvngoscope consists of a battery handle upon which is a demountable laryngoscope, provided in three sizes. Each laryngoscope contains a large lateral channel for the removal of intubation tubes, a large lamp situated as near the tip as possible; the whole a sterilizable unit which can be boiled, providing electric connections which are free from loose wiring. This equipment may be counted upon to function when needed. I have carried around one of these laryngoscopes in a handbag without protection, and have used only two or three lamps in the course of a year of almost continuous use. We have, therefore, a means of inspecting our field under conditions where resuscitation is required; namely, in asphyxia and under complete anesthesia. Under these conditions, we have practically a cadaver. Any man, properly instructed, can promptly laryngoscope a patient under these conditions. Fortunately, where a patient needs resuscitation most, we have the easiest possible conditions for laryngoscopy. I was drawn to this particular question by the necessity for some sort of scientific method for the resuscitation of new-born babies. The present treatment seems to be to let the youngster alone. It used to be the proper thing to spank them, and to put them into hot or cold water, or to try some other means of local irritation. But the technique now seems to be to let the baby alone. Usually it will breathe in due time, but once in a while it fails to do so. Some weeks ago I heard of a case—a new-born baby, who seemed to be doing perfectly well. It was put to one side, and when the nurse came to pick it up, it was dead. Another baby was put to breast; it seemed rather quiet. After a time, when the nurse came to put it back in its crib, it was found to be dead. Apparently asphyxiated. Both of these cases were susceptible to scientific medical treatment, based upon inspection, aspiration and insufflation. It is not only possible, but entirely practical, to use this instrument in the examination and treatment of new-born babies.

The next point in resuscitation, aspiration, is the removal of all foreign matter, chiefly fluid, in the respiratory airway. In the new-

born, amniotic fluid, meconium, etc. In the adult, vomited fluid, such as gastric contents, or intestinal fluid, as in the case of intestinal obstruction.

As electrical and water suction apparatus sometimes fails to work, we have assembled for use a 4-ounce bottle with a suction bulb and a special curved tip, which will empty a child's airway in three or four sucks. The operator exposes the pharynx and larynx when he sees the fluid to be aspirated, he removes it, securing, at once, a clear airway.

Following inspection and aspiration, we must carry out the practice of insufflation. Dr. Yandell Henderson, of Yale University, has brought out upon many occasions the life saving importance of carbon dioxid combined with oxygen in cases of respiratory failure. Dr. Henderson maintains that carbon dioxid is the normal respiratory stimulant; that this is constantly present in the circulation, exerting its beneficent effects. Whenever the respiratory center is depressed from loss of oxygen, as occurs in asphyxia, or from other conditions; infection, for example; the respiratory center loses its irritability and fails to respond to the carbon dioxid present. An artificial increase in the percentage of carbon dioxid increases the power of this respiratory stimulant and results in bringing about a respiratory effort. It must be remembered that carbon dioxid has nothing to do with the patient's color, and that acute asphyxia may be present with a low percentage of carbon dioxid.

In order to bring about the insufflation which is required, provided a field which is perfectly free, all that we require is some sort of tube to pass between the vocal cords into the trachea, and some sort of apparatus to give oxygen under pressure, and lastly, some means of controlling the pressure delivered. The pressure required for an adult will vary from 30 to 50 m.m. of mercury. In a new-born baby, the lung will expand and by auscultation one may hear air going into the axillary spaces with 15 m.m. of pressure. We tried in the beginning to keep the method as simple as possible, using a tube plunged into a large test tube of water, water being 13 times more sensitive than mercury. We first used an oxygen tank, such as one finds standing on the operating floor. We found that this low pressure tank was usually empty. We were forced, therefore, to use a small high pressure tank, necessitating a reducing valve. Some sort of holder was also necessary for the manometer, and it was found convenient to fasten this to a reducing valve. The apparatus before you is therefore relatively very simple. The con'ainer serves not only as a holder for the gas and manometer, but as a table as well.

For adult treatment, we have utilized the ordinary bronchoscopic tube, shortened to such a length that it will not touch the corina. In the new-born baby, we use a very small tube—4 m.m. While this tube appears large and almost threatens to traumatize a newborn baby, it will easily slip into the glottis of an unconscious or

asphyxiated baby, with plenty of room to spare.

A few words may be said relative to intratracheal anesthesia. This reference will be amplified by the motion pictures which are to follow. You are all familiar with intratracheal insufflation anesthesia. Intratracheal anesthesia of this type is customarily given by the anesthetic vapor being driven into the trachea by an electrical vaporizer, through a small silk-woven catheter, which allows the return flow around the tube. Magill, of London, added a return flow tube incorporated into the insufflation tube. The use of insufflation methods imply electrical apparatus, a manometer, and the complications which go with these conveniences. After observing the Magill two-way tube, it seemed that if one could use a tube that the patient could breathe through by his own respiratory efforts, doing away with complicated apparatus, this result was much to be desired. We have built up a tube consisting of a piece of bronchoscope. There are three sizes, 5, 7 and 9, corresponding with the typical Jackson bronchoscope. This piece of bronchoscope has been continued by a tightly-woven wire spring covered with rubber drainage tube, resulting in a tube which is both rigid and flexible in those positions requiring these possibilities most. The tube is intubated under direct inspection, and from the time of intubation we have practically no trouble with the patient. There is no post-operative aphonia or unpleasant effects from the use of the tube. I am using this as a routine for all operations about the head and neck. We have changed our operating field from a supratracheal to an extratracheal one. The method is available to anyone who wishes to take the trouble to acquire the simple technique of laryngoscopy in the anesthetized patient. For a more complete discussion of this subject, you are referred to the "Archives of Otolaryngology", February, 1928, and the Journal of the American Medical Association, Sept. 15, 1928.

1819 Broadway.

A FURTHER STUDY OF THE RELATION BETWEEN THYROTOXICOSIS AND TONSILLAR INFECTION.

Dr. Louis E. Brown, Akron, Ohio.

In a paper presented before the Colorado Congress of Eye, Ear, Nose and Throat surgeons, in 1920, the author called attention to the possibility of infected tonsils as a causal factor in the production of some types of goitre.

I intimated at that time the possibility of a greater percentage of goitres being of toxic origin than had previously been supposed and suggested that infected tonsils might be one of the principal foci, a suggestion which since has become, according to clinical findings, a well founded fact.

In 1922 I submitted an article on, "The Relation Between Thyrotoxicosis and Tonsillar Infection", advancing the theory that many of the goitres seen were of toxic type and that infected tonsils played an important role in a great many cases, and in some were the direct causal factor. This of course was a preliminary report and based on a comparatively small number of cases. It has been interesting and instructive to watch the progress and study which has been given this subject since the above article. Surgeons and physicians alike are recognizing toxic goitre, where it had not been recognized before and thus making it almost a definite entity.

In 1922 Dr. Graham objected to the term "toxic goitre" as indefinite and subject to various interpretations. However, I believe this objection has been almost completely removed, for only recently did Dr. Graham lectured on goitres and gave a very excellent differentiation between the toxic type and the endemic type.

He heartily concurs with the author in believing that infected tonsils are a definite foci, and should always be removed in all cases coming under this heading. In a study of a group of 50 cases coming under his observation, 30 were of the toxic type and required both thyroidectomy and tonsillectomy to relieve all symptoms.

To date there have been several articles and lectures given propounding the same theory as that advanced by the author. So much evidence has accumulated, in fact, that it is reasonable to say that today it is a known fact that this relationship does exist and that tonsils are a definite causal factor in many cases of toxic goitre.

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Having observed a greater number of cases and over a longer period of time, I am quite convinced that this syndrome is assuming the role of a distinct entity and claims the attention of and closer co-operation between physician or surgeon and the laryngologist.

It has been my experience to see many of these patients and call to their attention the fact that they were developing a goitre, which, after a careful history, examination, metabolism test and observation, proved to be of the toxic type, and greatly influenced by an attack of tonsillitis or peritonsillar abscess. Some of these cases have received a great deal of benefit and shown marked improvement following a tonsillectomy and the administration of iodin in some form, preferably Lugol's solution. Most of them, however, had progressed to such a point that thyroidectomy was necessary, and in these cases it was found necessary to do a tonsillectomy either as a preliminary operation or as soon after as consistent, in order to obtain complete relief from all the disturbing symptoms occurring in these cases.

After all these years we are still unable to state definitely what the modus operandi is in the transmission of the toxins from the infected tonsils to the thyroid gland, neither have we been able to prove by culture or chemical tests that this is a fact. So far we have been compelled to rely on history, observation and clinical findings, but it is my hope that by further study of this subject we will arrive at some definite conclusion in reference to this point.

Having recognized that there is a division in the type of enlargement of the thyroid gland, known as toxic goitre, what are the most probable sources of foci from which the toxine may be derived that will produce this hypertrophy? May it not be the infected tonsils? It might also be well to consider the sinuses and teeth when making this survey. However, up to the present time the author has not seen a case which he could attribute to either of the last-named sources. A further study of this source of infection is necessary.

Some theories which the author would like to advance regarding the changes that take place within the gland and certain factors which may act as a probable cause of this condition are as follows:

First, may it not be possible that in a certain number of cases due to the deficiency of the iodin content in the thyroid that its resistance to certain types of infection which may have a definite affinity for the thyroid, the toxine of which is generated in the tonsils, might explain this syndrome?

Second, is it possible that the infection which migrates to the thyroid gland may have a selectivity for certain structures of the thyroid and thereby inhibit their action in the production of thyroxin and thus bring about the change that occurs in the parenchyma of the gland, for it is a well known fact that these changes do occur where there is some interference with the iodin content of the gland?

Third, what is the status of the lymphatic system in patients suffering from a toxic goitre? This should be taken into consideration and studied. The surface has only been scratched in the study of this most important phase of the subject.

Fourth, what percentage of goitres are developing among the people, particularly girls nearing the adolescent age, where the tonsils and adenoids have been removed, compared to those who have not had a T. & A. done? This is an interesting point and observations over a period of five to 10 years—particularly among the girls—would furnish some interesting and illuminating data on this subject.

Fifth, is it not possible that some individuals have certain areas that are congenitally less resistant than is normal, and that such areas are the ones to show evidence of disease, such as has been described above, namely, toxic goitre?

Sixth, a very interesting experiment that might be carried out in a goitre area would be to take a group of patients with a beginning goitre, remove their tonsils and feed them on a well balanced diet consisting of vegetables and fruits produced on soil which had received special attention regarding the iodin content of same by having this garden sprayed regularly with water specially prepared with iodin, and note the results over a period of months or years.

Having advanced the above theories, to substantiate them, I wish to call the attention of the profession to some of the work that has already been done.

The theories advanced in an article by Dr. H. T. Bailey, of Phoenix, Ariz., are that "the thyroid gland is a manufacturing plant and a storehouse of colloidal material, which is iodin connected to protein, forming a compound called thyroglobulin, the active principle of which is thyroxin. This colloidal material is taken out of the storehouse by the lymphatics and circulation and carried to the part of the body where it is needed most; for example, to the nervous system, the vascular system, skin, hair, sexual organs, the osseous system, etc. If there is too much or too little colloidal material manufactured by the gland there will be too much or too little stored in the storehouse; therefore there will be too much or too little absorbed and carried into the system, thus there will be alterations in the aforesaid structures, either as an over-stimulation or under-stimulation. It matters not what stimulates the thyroid gland to an over-production of colloidal material, it causes a thyrotoxicosis, and if we have an under-

stimulation we have cretinism in the child and myxedema in the adult."

And to verify his findings in this group of toxic goitre cases, he cited several definite cases in which the patient had a very acute exacerbation of all the cardinal symptoms, with a marked temporary enlargement of the gland following an acute tonsillitis and was relieved only after thyroidectomy and tonsillectomy were done.

Dr. Squier and Dr. Newburg, of the University of Michigan, say that "in those cases of Graves' disease which have come under their observation, the appearance of an acute infection very commonly has led during the course of the infection to an exacerbation of all symptoms of thyroid disease. Accordingly, the search for and elimination of focal infection has become an accepted routine in the medical treatment of this condition, and the improvement seen following the removal of such foci has frequently been striking."

Whether or not the thyroid itself is actually invaded during infection can only be conjectured. However, it is quite possible that the infection does reach the gland, sets up an acute nonsuppurative thyroiditis which destroys the cells and leaves scars throughout the gland, followed by hypertrophy of the other tissues.

This may be the result of the action of certain bacteria that produce an extracellular toxin which, as stated above, attacks the cellular structure of the gland and thus prepares the soil by breaking down the normal barriers of resistance, making it very easy for the establishment of a chronic infection, followed by hypertrophy. This may also be explained by the phenomena that certain bacteria seem to have become so fixed in their habits as to produce, when they attack the organism, diseases which, while they vary in each individual case in intensity and minor symptoms, are easily recognized as definite entities. While still other bacteria seem capable of producing, under circumstances which we cannot as yet classify, infectious processes of either acute or chronic form, the manifestations of which are so diverse that their recognition as a definite disease process is impossible.

Roger and Garnier's investigations apparently attracted little attention in this country, and it was not until Billings, Rosenow and their co-workers began to publish the results of their researches on focal infection, that the possible relation of the thyroid to systemic disturbances was brought to general notice in the United States. In his address on focal infection, delivered before the American Medical Association in 1914, Dr. Frank Billings reported three cases of rheumatism attended by acute tonsillitis and thyroiditis, attributing all

three manifestations to a single focal cause. He went on to say that the interest of his clinic was aroused to the possibility of focal infection as a cause of goitre. He reported seven additional cases which "seemed to show that there is an infectious type of goitre with and without symptoms of exophthalmic goitre, which seems to be of toxic origin. The rapid subsidence of the goitre and the symptoms after the removal of the foci of infection in the jaws and tonsils was a surprise." All of these patients gave a history of chronic tonsillitis. Dr. Billings ended by saving that to the list of acute conditions already known to be due chiefly to focal infection, we were now justified in adding several others, including "certain infectious types of thyroiditis, with or without hyperthyroidism". A year before Dr. Billings' address was made, Dr. Clement F. Theisen, of Albany, N. Y., published some cases of acute tonsillitis complicated by an acute thyroiditis. These cases offer perhaps the best support which the present writer has been able to find in the literature of a close inter-relation between infections of the tonsil and the thyroid. Theisen believed his cases to be of "particular interest from an etiological standpoint, as in all except one case, the inflammation of the thyroid gland occurred with or directly after attacks of tonsillitis. Two of these patients have each had two distinct attacks of acute thyroiditis, each time with an acute tonsillitis, and both have since developed well marked diffuse goitres." While Theisen does not wish to be understood as emphasizing the foregoing facts as important etiological factors in the development of goitre, he feels that it is by no means impossible that the repeated inflammatory attacks to which the gland was subjected may have, partly at least, been responsible for the subsequent chronic hypertrophy of the thyroid gland.

If we consider now, briefly, the function of the thyroid and its secretions, we can, perhaps, point out how these chronic foci cause those changes in the thyroid which result in the pathological condition generally recognized as goitre.

The exact biochemical function of the thyroid and its secretion is even now little understood, despite the vast amount of experimental work that has been done. The utmost difficulty surrounds the obtaining of the secretion of any ductless gland. No analysis of the gland juices obtained by pressure from the removed gland or the gland as a whole can be presumed to represent its real internal secretion. This secretion may only reach its final form as it enters the blood stream, or it may be changed immediately with its admixture with the blood. There is little doubt, however, that the secretion contains constantly an iodized albuminate, the iodothyrine of Bau-

mann, or thyroglobulins. The presence of iodin seems to be constant, but in varying amounts. Thyroid secretion seems to act after entering the blood stream as a biochemical agent of an oxidizing nature, regulating metabolism, a process which it probably accomplishes through its control over the vagus nervous system. But in whatever way this control is carried out, it seems to be dependent upon a constant iodin content in the secretion.

The importance of this iodin content of the thyroid secretion was very carefully studied and brought out in an article by Dr. Judson Quimby in the New York Medical Journal a few years ago, but time will not permit a review of it here. However, it seems possible that under its influence the tissues by which nutrition is activated are in a better condition to efficiently oppose and defend themselves against any infection or toxine which may attack them.

It is interesting to note that as far back as 1900 Paul Bourcat recognized the fact and importance of the constant fight which must be waged by the thyroid secretion against bacterial invasion and its associated toxemia.

In further support of the author's hypothesis of the relationship existing between infected tonsils and toxic goitre, he begs to quote at length from a paper by Dr. S. P. Beebe, of New York. He says that "from time to time he has put himself on record as one of the strong supporters of the theory of infective inter-relation between the tonsils and the thyroid. He calls attention to numerous clinical observations on the relation of thyroid disease to previous infections. In this connection, it is well to remember that thyroid disturbances occur most frequently in persons of a thymolymphatic constitution, and it is these individuals who are most susceptible to infections. The terminal event in hypertrophied patients is not infrequently an infection which has begun in the tonsil. A large percentage of patients with exophthalmic goitre have enlarged tonsils and adenoids. from a particularly severe attack of tonsillar infection. Infections It is not uncommon to date the beginning of a thyroid enlargement in the nose and throat are undoubtedly the most common to which the human family is subjected, and the tonsil is one of the most important points of entry we have for infections, but in goitre the resultant condition is a hyperactivity of a gland of internal secretion and not a continued infection. It is obviously more difficult to explain such a result than to trace the connection between an acute tonsillitis and a septicemia, or an infected joint.

Infection does not in a large percentage of cases produce such an enlargement of the thyroid gland that it would be recognized as a goitre, and it may be that the thyroid does not react in this manner

except in those who are not quite normal in respect to the balance of their glands of normal secretion. If the thyroid secretion is an important element in the defense against infections, it is not impossible that it is stimulated to over-activity when occasion demands, and if the stimulus be often repeated it may lead to changes which we recognize as pathologic. Through the repeated stimulus to overactivity, the gland has become hypertrophied and its heightened function continues long beyond the stimulus which originally calls it forth.

Clinically, there is an important relation between the infections in the nose and throat and hyperthyroidism. In patients between the ages of 16 and 24, from 35 to 40 per cent gave a history of repeated attacks of acute tonsillitis and a goodly percentage of these have enlarged thyroids. Rapid enlargements of the thyroid, with characteristic symptoms of over-activity, have often followed immediately after a particularly severe tonsillar infection. Such patients bear these infections badly. Their convalescence is slow, and each attack is accompanied by severe prostrations quite out of proportion to the apparent severity of the infection. Dr. Beebe has observed that the leukocytosis in these cases is lower than that of nongoiterous patients and that hyperthyroid patients often show a marked leukopenia with a relative lymphocytosis, indicating some influence on the blood picture operating when the organism is subjected to infection.

The tonsil infections to which exophthalmic patients are so often subject, constitute most dangerous and distressing complications, and the alert surgeon should always be on his guard against them. If there is active thyroid intoxication it is seldom wise to enucleate tonsils and adenoids, because such patients react badly to operations of any sort. The anoci-association methods so successfully employed by Crile in thyroidectomy, which Beebe described as "stealing the thyroid", ought always to be employed in all operative measures on cases of this type.

The author has felt justified in quoting thus at length from Dr. Beebe's excellent paper, as the position he occupies is in most respects analogous to the writers' own, and the opinions brought out in the discussion which followed the presentation of the paper before the Laryngeal Section of the American Medical Association, very closely resembles those held by the writer and by many of the men to whom the author sent questionnaires covering this subject:

The great mistake that has been made in the past, is that tonsillar and closely related nose and throat infections have been considered

in this etiological relation to the almost complete exclusion of all other sites of chronic infection. That is not the purpose of this paper, but rather to point out the amazing relation that these foci of infection bear to thyroid changes, and to suggest that in time we may find that the type of goitre is merely the response of a certain individual thyroid to the character and type of infection it is called upon to combat. A focus of infection, be it situated where it may, is a damaging factor in the health of anyone carrying such a focus. The amount of harm resulting therefrom varies in each case, and is most frequently not at all in direct proportion to the local manifestations at the site of the foci. This variation in the systemic manifestations depends on:

First: The type and virulence of the infecting organism.

Second: The normal resistance of the individual to the type of infection, the normal amount of activity of the protective functions of the blood and thyroid function. These differ greatly in individuals, as has been shown by the researches of Hastings and others.

Third: The degree of loss of this resistance under mental and physical stress and strain.

It is interesting how many individuals will carry about one or more areas of infection and for years, under normal living, free from worry, they have little or no systemic symptoms, but as soon as these same individuals undergo some sudden or severe physical or mental stress, the systemic disturbances become marked. This merely means that the load has become too great, and the protective forces of the organism fail. These cases suddenly develop one or more of a variety of pathological conditions, depending on the area affected by the bacteria or their toxines.

It would give the author great pleasure to continue to quote at length the answers he received confirming his theories and deductions concerning this most important subject; these were in response to the many questionnaires sent out; but time will not permit.

However, suffice to say, it would seem that the consensus of opinion to date concurs with that of the author; however, much research and study must be done on this most interesting and significant subject before any definite conclusions can be stated and it is with that thought in mind that I again present this subject for your consideration, hoping that it will stimulate interest and constructive criticism and a more widespread consideration of the relationship that probably exists between infected tonsils and thyrotoxicosis.

SUMMARY.

First: It is surprising how little consideration has been given to this most important subject.

Second: That toxic goitre is practically a clinical entity today.

Third: That as yet the *modus operandi* in the transmission of the infection to the thyroid is not clearly established.

Fourth: That there are numerous experiments which might be carried out in an attempt to prove or disprove the theories advanced concerning this relationship.

Fifth: That it is not the purpose of this paper to infer that infected tonsils are the exclusive source of infection, but rather to point out the possibility of them being one of the principal foci and to urge closer study of any cases that may appear to be of the toxic type of goitre.

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A REFINEMENT IN THE TECHNIQUE OF INTRABRONCHIAL INJECTIONS.

DR. MILTON S. LLOYD, Staten Island, N. Y.

So many methods have been devised, especially since the creation of lipiodol, for intrabronchial injections that it may seem useless, not to say impertinent, to add another technique to a field already sufficiently encumbered. The writer believes, however, that the method to be described combines most of the advantages of present practice, while it obviates many weaknesses, and is therefore worthy of presentation to the profession.

The apparatus consists of: a. a soft rubber catheter (No. 19, French) cut off to the length of 36 c.m., into the open (squared) end of which is inserted an adaptor for a Luer syringe; b. a pliable wire, which is provided, 35 c.m. from its end, with an enlargement suited to the adaptor in the catheter and beyond which the wire is shaped into a ring for ease of manipulation; c. a 20 c.c. Luer syringe. The free end of the wire is bent into the usual curve of a laryngeal syringe and inserted into the catheter, until the adaptor is closed. The whole is then ready for use.

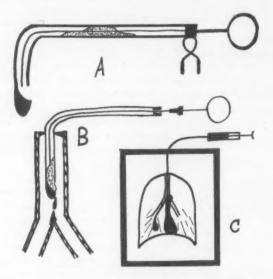
To make an injection, the adaptor of the catheter is uncorked and about 1 c.c. of 5 per cent cocain is dropped into it. The catheter is recorked and can then be held in any position without losing the anesthetic. A few drops of cocain are allowed to fall onto the vocal cords, or a wet applicator is rubbed over the cords and the posterior surface of the epiglottis. Under the guidance of a laryngeal mirror, the curved catheter is then inserted into the larvnx and if the patient coughs the free end is raised and uncorked, allowing the contained anesthetic to escape into the trachea. In a few moments the cough reflex is subdued. If there is no cough, the free end of the catheter is lowered and the cocain is allowed to escape from the open end. The catheter is then pushed forward along the shaft of the wire and sinks deeper into the trachea. While this is being done, the patient is asked to lean rather sharply toward the side in which the injection is to be made. The tube will then enter the corresponding bronchus. In the erect position it will nearly always enter the right bronchus. As the free end of the tube approaches the lips, it is held in position by the left hand, while with the right hand the wire is drawn at first

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downward and outward, then upward and to the right. As the hand nears the right ear, the end of the wire will emerge from the tube. The catheter is then attached to the lip, either with a special clip or with a piece of adhesive tape. The patient is conducted to the fluoroscopic room.

The syringe is filled with the warmed lipiodol, other medication, and its tip is inserted into the adaptor of the catheter.

On the fluoroscopic screen, the position of the tube is generally apparent. If it is not, it can easily be rendered so by the injection of lipiodol, until a few drops escape from the end. If the tube is



in the desired bronchus, the proper quantity of liquid is injected under fluoroscopic control. It may be changed to the opposite side by simply asking the patient to lean in that direction, withdrawing the tube and reinserting it.

After the injection the tube is removed by pulling on the free end. The accompanying diagrams explain the apparatus and its use: Fig. A shows the catheter mounted and ready for use. Fig. B. shows the tube inserted into the larynx, the adaptor uncorked and the cocain escaping into the trachea. Fig. C shows schematically the view in the fluoroscopic screen. The tube is in the right bronchus and sufficient lipiodol has been injected to fill one bronchiectatic

cavity at the base. The proximal end of the tube and the syringe are shown for clearness.

The method has the following advantages:

- 1. It is simple.
- 2. By avoiding the nasal route for the catheter, a minimum amount of anesthetic is used and there is no danger of wiping infective material or crusts down into the bronchi.
- 3. The danger of allowing part or all of an injection of lipiodol to enter the stomach is obviated, and the risk of iodism is reduced to a minimum.



- 4. No needling or penetration of the tracheal wall is necessary.
- 5. The injection is made under fluoroscopic control and may be unilateral if desired. At any rate, neither too much nor too little of the liquid need be used.
- 6. While not giving the information offered by direct observation through the bronchoscope, the method is simpler; may be more universally applied; and the apparatus may be made for a trial at least by anyone who has a mechanical turn of mind.

Sea View Hospital.

International Digest of Current Otolaryngology.

Editor.

DR. MAXWELL FINEBERG. St. Louis.

Collaborators:

Mr. W. S. Daggett, London. Priv. Doz. Dr. G. Keleman, Budapest. Dr. D. E. Staunton Wishart, Toronto. St. Louis Jewish Hospital E.N.T. Journal Club.

The Thirty-fourth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology will be held in Atlantic City, Oct. 21-25, 1929, with headquarters at Hotel Traymore.

We are pleased to publish verbatim the Foreword of the program issued by the president.

FOREWORD.

A perusal of this program, arranged for the Atlantic City meeting, reflects careful planning and much work by the Program Committee.

Aside from the scientific assembly, which bids fair to be worth while, many important changes in the government of the Academy will be presented for final action, and every member of this great body who can be present is urged to familiarize himself with the proposed changes ,that deliberate and careful judgment may be exercised.

Special effort has been exerted to make the banquet a fitting climax of a splendid meeting—it is the earnest desire of the committee that all members and guests attend. Dr. Harris P. Mosher, Pres.

The guest of honor of the Academy is Herbert Tilly, M.D., B. S., F. R. C. S., Senior Surgeon, Royal Ear and Throat Hospital, University College Hospital, London. His address will be on Tuesday morning, Oct. 22, at 9:00 o'clock, on the subject of "Some Experiences in the Surgical Treatment of Inflammation of the Frontal Sinuses and Possible Complications".

A very attractive schedule of conferences has been arranged both in Ophthalmology and Otolaryngology, a list of which can be obtained from the secretary of the Academy.

The scientific program is arranged in a very attractive form and among the numerous papers is a symposium on Brain Abscess and a symposium on the Ethmoid Region.

The social aspect of the meeting has not been neglected and many interesting features have been arranged.

The examinations of the American Board will be held in Philadelphia on Oct. 21.

Any further information may be obtained from the secretary of the Academy, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb.

Coryllos, in the American Journal of Medical Sciences, July, 1929, reports on some further observations on bronchoscopic examinations in lobar pneumonia. He aspirated the occluding exudate and found that the mortality was markedly lessened. He is as yet unwilling to state that this is absolutely due to the bronchoscopy as he has not done a sufficient number of cases. The work, however, does prove that a carefully performed bronchoscopy is not hazardous in a case of pneumonia.

M. F.

Gibbs, of Halifax, in the Journal of Pharmacology and Experimental Therapeutics, Baltimore, June, 1929, reports on the effect of ceanothyn extract on blood-clotting time. He states that ceanothyn extract taken by mouth in quantities up to 80 c.c. could not be shown to alter the normal clotting time.

M. F.

Yearsley, in the British Journal of Children's Diseases, London, April, 1929, reports three cases of deafness in children which were due to a chronic intestinal intoxication. He claims he has often found chronic intestinal intoxication associated with septic tonsils and carious teeth. He concludes that chronic intestinal intoxication is the primary septic focus.

M. F.

Dr. Arthur C. Jones, of Boise, was elected president at the Seventeenth Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, held at Salt Lake City, July 2.

Thompson, of Fort Worth, in the June, 1929, Annals of Otology, Rhinology and Laryngology, reports the use of a small rubber drainage tube in the abortive treatment of peritonsillar inflammation. He does not wait for the abscess to form and point but makes his incision even in cases where the inflammation is as yet slight. He spreads the incision in the usual way and then inserts a small piece of catheter tubing and leaves it in siture. He winds a thin layer of adhesive plaster about the proximal end of the tube. Thompson claims that by means of this treatment he has been able to save his patients many days of starvation and suffering.

At the Annual Meeting of the American Bronchoscopic Society, held in San Francisco, in July, Dr. Thomas Carmody, Denver, was elected president, and Dr. Louis H. Clerf, of Philadelphia, secretary for the ensuing year.

Dr. H. P. Mosher, of Boston, has been tendered the honor of giving the 1929 Semon Lecture of the University of London. His address, entitled, "The Lower End of the Esophagus at Birth and in the Adult", will be delivered in the Hall of the Royal Society of Medicine, London, Dec. 5, 1929.

Trotter, of Buffalo, in the June, 1929, Annals of Otology, Rhinology and Laryngology, presents an article on the use of nerve-block in tonsillectomy. Some of the advantages claimed are complete anesthesia during operation, lessened surgical shock, complete postoperative function of the muscles of deglutition (lessened possibility of aspiration pneumonia), anesthesia lasts some few days, thereby lessening post-operative discomfort. Trotter uses quinin and urea hydrochlorid, one-half of 1 per cent in strength, and injects 1 c.cm. The technique of his nerve-block is based on the anatomical fact that the nerve supply of the tonsil reaches the tonsil through a little muscle called the tonsillopharyngeus. This muscle can be reached by means of a straight needle through the anterior pillar just skirting the capsule of the tonsil until a resistance point is met. This resistance is the tonsillopharyngeus muscle. It is most easily reached by placing the needle about half a centimeter lateral to the edge of the anterior pillar and about 1 cm. above the inferior pole.

Trotter has used this method in 126 cases and is enthusiastic about his results.

M. F.

The Clinical Congress of the American College of Surgeons meets for its Nineteenth Annual Session in Chicago, Oct. 14-18. Major-General Merritte W. Ireland, Surgeon-General of the United States Army, will deliver the inaugural address. On the program are such names as Professor D. P. D. Wilkie, of Edinburgh; Dr. Glenn Frank, President of the University of Wisconsin, and Mr. Herbert Tilley, London. The complete program will be found in the September issue of Surgery, Gynecology and Obstetrics.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

March 8, 1929.

Inner Ear and Cerebellar Changes in Pathologic Fetal Position; Their Significance for Static Disturbance in Earliest Periods of Life and for Some Anomalies of Speech. Prof. Antonin Precechtel.

(Published in full in the July, 1929, issue.)

SECTION OF LARYNGOLOGY AND RHINOLOGY.

March 27, 1929.

DR. HURD (Past Chairman): I thank the Chairman for the compliment of this honor. I was chairman in 1911, so long ago that I have forgotten all about it. We had pretty good audiences then, though the room was small. Owing to limited time, I think we better proceed with the program.

DISCUSSION.

DR. W. W. CARTER: I am especially interested in one of these cases that Dr. Dunning has reported. I recall a similar case of destruction of the ala which I corrected in a different way from the method he recommends. My patient was a woman of 25 years, very good looking, who had suffered the loss of her right ala. She had had a small growth on the edge of the ala, supposed to be a hematoma, and was told that it could be removed by the X-rays. These were employed and destroyed the ala. She did not want me to take a flap from the face, for she did not wish an additional scar, and I thought of something rather radical. I told her the possibilities were 1:10 though of something father that the result would be successful. I made a pattern of the dehiscence in the ala and from that pattern I made another of adhesive plaster and pasted it on to her ear, selecting a portion of the ear which corresponded almost exactly to the ala, making the graft about one-third larger than the defect, thus allowing for subsequent contraction. After removing quite a little scar tissue from around the dehiscence which had ben caused by the X-rays, I introduced the graft into position, fitting it as exactly as possible. I was much afraid that I would lose it, for the transference of massive grafts is always attended by considerable hazard. I then used an appliance I devised some 20 years ago -a hot water jacket to keep the graft at the proper temperature and give it opportunity to unite. This appliance is attached to a douche bag and the water is put in at a temperature of 101° and is about 100° when it reaches the graft. In that way the graft was kept quite warm for three or four days after being put in position. It took perfectly, and the young woman had complete restoration of the ala. She was in to see me a few days since, and you could hardly tell which side had been operated on 18 months ago. I had carefully brought together the edges of the wound in the ear, and one ear is a bit smaller than the other, but that is not noticeable with the present mode of dressing the hair. I think the best definition I ever heard of a young lady's ear is that it is something not to be seen. I regard this method as ideal for the correction of such a defect if we are so fortunate as to be able to keep the graft alive until it unites. This hot water splint was presented before this Section and also before the Trilogical Society about 15 years ago, but I notice that one of the instrument-makers is now attributing it to someone else.

(Replying to Dr. Dunning): I took the full thickness of the ear so that the skin was on the inside as well as on the outside.

Exophthalmos Due to Chronic Abscess of Orbit and Chronic Frontal Sinusitis. Dr. C. G. Coakley.

> (To appear in a subsequent issue of THE LARYNGOSCOPE.) DISCUSSION.

DR. T. J. HARRIS: I have nothing to say except to congratulate Dr. Coakley on the success secured in this most difficult and baffling case, particularly as regards the diagnosis. The case has many unusual features and I am not surprised that he was unable to make the diagnosis at the outset; but the

results secured are worthy of very high commendation.

DR. COAKLEY: There is nothing to note except the unusual position of the swelling; the case was not like an ordinary infection. I had a similar case 17 years ago and the patient got well after two operations; the same mistake was made in each instance, of leaving a portion of the membrane way back, making it necessary to go in a second time and remove it. We hesitated to do this at the first operation on account of the condition of the patient, and hoped it would be absorbed, but it was not.

Recurrent Tracheal Stricture. Dr. J. D. Kernan.

(To appear in a subsequent issue of THE LARYNGOSCOPE.) DISCUSSION.

DR. C. G. COAKLEY: I saw this patient in 1916, and sent her to the hospital, where she was tracheotomized by a general surgeon, and she got along very nicely. I did not see her again until last October, when I was at the hospital and was told that a patient wanted to see me, and I found that one of our assistants had done an emergency tracheotomy, as she was almost in extremis; she was just recovering from a pneumonia and was cyanosed and having oxygen, but came through a little later. The amount of secretion that was coming up from the tracheal wound was something astonishing. She is very lucky indeed to have recovered. It is a most unusual case of stricture progressing down the trachea.

Recurrent Polypi of Maxillary Sinus in a Boy of Eight Years. Dr. J. W. Babcock.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)
Dilatation of Esophagus with Spasmodic Stricture. Dr. George R. Brighton.

> (To appear in a subsequent issue of THE LARYNGOSCOPE.) DISCUSSION.

DR. C. J. IMPERATORI: I have seen two similar cases, that I reported as megaloesophagus. In my opinion, this dilatation will continue and there is no way of prematurely relieving it. It is very likely the Roentgenologist will always be able to identify this particular case. That is, this esophagus, as it is today, will practically remain that way. Repeated Roentgenographs have shown this observation. Dr. Jackson made this observation some years ago and called attention to it, but apparently little heed was taken of it.

In my opinion, this is not a case of cardiospasm, preventriculosis, phrenospasm, achalasia or any other term one desires to call this condition. It is true that in the above mentioned condition, that is, cardiospasm, there is a dilatation of the esophagus due to spasm, or, better, due to spasmodic occlusion

of the lower end of the esophagus.

It is probable that this woman had an ulcer at one time. I would suggest to Dr. Waugh that he use a Mosher hallooning esophagoscope. In this way, he will be better able to follow the wall of the esophagus. Using a thread as a guide in cases of large dilatation of the esophagus assists materially in in-

specting the mucosa.

DR. J. M. WAUGH: I had no intention of joining in the discussion, but it is my impression that it is a case of congenital megaloesophagus; they do not give symptoms for a long time, but in the course of time there is a sagging of the esophagus, which produces the kinking. I don't think the spasm is the primary cause; they will have spasm at times, but not a real spasm, the kinking produces it; and the reason they do not get the benefit from lavage the tube goes down into the area. I have seen the case of a man who had the trouble for years; he had considerable low-grade ulceration, and it is almost impossible to examine the wall thoroughly with the esophagoscope, for it is collapsed when you do it; and there is an enormous dilatation and you get just such a picture as Dr. Brighton showed. When you do get a chance to examine these patients, the physical symptoms do not make the diagnosis; you get that with the X-rays. The patient will go on for a long time, but it is questionable whether it is a primary spasm; I don't believe a primary spasm will ever produce that. I think it is analogous to megaloesophagus in children. It is a very interesting case.

DR. BRIGHTON: We felt very much as Dr. Waugh does in this case. We started out by calling it a megaloesophagus, but felt hesitant, so we called it a dilatated esophagus and waited to see what would happen. There was a spasm and stricture there, but which came first we were not prepared to say.

Report of a Case of Composite Tumor of the Tonsil. Dr. Geo. V. Browne.

(To appear in a subsequent issue of The Laryngoscope.)

Lymphosarcoma of the Tonsil. Dr. Geo. V. Browne.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DR. C. G. COAKLEY: I have seen two cases like the pharynx case, and in one the mass was seen on the right side and extended up to the soft palate—so much so that it wa salmost impossible for the patient to swallow. These mixed tumors are not very malignant and very seldom have involvement, and when removed either do not come back at all or very rarely. The size is often interesting. All that I have seen could be reached by intrabuccal operation

instead of from the outside.

Dr. C. J. Imperatori: I have recently seen the case of a well known physician, who had a lymphosarcoma of the tonsil. I referred him to Dr. Wood, who in turn sent him to the General Memorial Hospital, and there a radium pack was placed on the outside, adjacent to the tonsil. I saw him on Feb. 15 with this enormous tonsil and again yesterday. The diagnosis by Dr. Ewing and Dr. MacNeal was that of lymphosarcoma. One would wonder what had become of this mass of tissue. My experience has been that the use of radium may do some good, and it apparently does do good in those cases that you can approach, but that usually in a very short time the patient has a general sarcomatosis. Whether or not the condition is disseminated by the radium, I do not know, but I do know that all these patients lose much weight, become apparently cured of his tonsillar growth.

DR. G. ALLEN ROBINSON: I am of the opinion the tumor in the tonsil which Dr. Browne reports as primary lymphosarcoma is really a metastasis from the embryonal carcinoma of the testis, which was removed a few months previously. The histologic picture of these two conditions often simulate each other. They are both radio-sensitive and metastasis usually occurs. We had recently, on Dr. McCullagh's service at the Manhattan Eye, Ear and Throat Hospital, a patient with a large growth in the nasopharynx with invasion of the cervical lymph nodes. Sections revealed a reticulum-celled sarcoma and was similar to a primary tumor that had been removed from the left ovary one

year before.

Dr. Browne: The diagnosis made by our pathologist was carcinoma in one case and lymphosarcoma in the other. I did not see the slides, for I would not be able to recognize them.

Presentation of Anatomical Specimens. Dr. Harry Neivert.

I have here a group of specimens, picked up from time to time in the laboratory and prepared several years ago for teaching purposes. This work was done in the laboratory of Prof. J. Parsons Shaeffer at Jefferson Medical College while a Fellow of the Rockefeller Foundation.

lege while a Fellow of the Rockefeller Foundation.

1. Sagital section of adult head, showing the complete Waldeyer's ring in an adult, a huge lingual tonsil branching off to the faucial, with a continuation up the lateral column, and a huge adenoid or pharyngeal tonsil.

2. A metal cast of the upper respiratory tract in situ, including the oropharynx, nasopharynx, nasal chambers, the paranasal sinuses, the Eustachian tubes, the mastoid cells, the lateral sinus, sigmoid, jugular bulb and jugular vein. It is the only specimen showing all these details made in one casting. The material used is Woods' metal.

3. This is a specimen of the frontal sinus. The entire roof of the orbit is excavated by ethmoidal cells. The superior and middle turbinates are completely excavated by a posterior ethmoidal cell. The sphenoid is unusually

large, extending down to the pterygoid process.

4. This specimen shows what at first appeared to be a complete absence of the antrum. The anterior limit of the antrum corresponds to a line between the posterior third of the inferior turbinate. The nasal wall of the antrum juts out, and on putting in a trochar cannula it would surely go through the cheek. There is no cavity at all in front of this line.

5. This is an unusually large sphenoid, showing the impression of the optic nerve, the three branches of the trigeminal nerve; the nerve to the pterygoid

canal (Vidian), the carotid artery, palatine ganglion.

6. This is an accidental specimen I picked up, with the mucous membrane removed from the superior turbinate, showing the distribution of the olfactory

7. Another huge sphenoid. In this specimen, the sphenoid sinus extended into the septum. Undoubtedly many of you have opened into the sphenoid, and this is what you found. I found many such specimens. Note the large extent of the sphenoid, the large septa, the three branches of the trigeminal nerve, and the carotid artery.

8. This specimen demonstrates the feasibility of irrigating the antrum over the inferior turbinate at the so-called undefended area of the antrum. You will note the uncinate process came down in a double projection to the inferior

turbinate.

9. This is an unusual specimen of a frontal sinus developing from a bullar cell. The entire frontal sinus on both sides was shown to have grown from the bullar region.

10. Another huge ethmoidal development, extending into the sphenoid and

almost completely displacing it.

11. This specimen shows the complexity of the ethmoid labyrinth, also the marked indentation of the second branch of the trigeminal into the sphenoid

Report of Cases of Nasal Accessory Sinus Disease. Dr. L. M. Hurd.

Mrs. I. M. D., age 52 years; 1919. Transillumination: Right antrum dark. Examination showed pus in right and left ethmoids, and sphenoids. Patient had headaches and purulent nasal discharge. Operation: Submucous and intra-nasal ethmoids, sphenoids and right antrum. Could not enter right frontal.

In 1920, Patient had pain about right frontal from 10 a. m. to 3 p. m. Some pus in nose. Entered right frontal intranasally and enlarged opening with rasp. Rather small anatomical limits and very hard bone.

1926: Ophthalmologist reported muscle unbalance right eye, pain over right e. Two months later slight swelling right frontal floor, mucocele suspected. Four months later, diplopia. One month later, swelling had increased. X-ray showed dense right frontal. Right ethmoids showed thickening of remaining walls, post-operatively. Right frontal transilluminated dark. Killian operation was performed. Three drams of very thick, gelatinous pus removed from upper inner part of orbit, there was erosion of entire bony floor of frontal. Cavity of frontal filled with granulations and polypoid tissue, which was removed. No opening into nose found, and a large opening was made by removing ante-

rior ethmoid walls and descending process of frontal bone. Smooth convalescence made. Patient remains well 2½ years after operation.

Case 2: Mrs. R. V. V. M., age 41 years. Several attacks of sore throat and coryza, with cough, hoarseness, hay fever, intense headaches, especially on the left side. For relief of headaches she had contracted the bromo-seltzer habit. X-ray showed both ethmoids cloudy, right antrum cloudy, left antrum contained growth, either polyp or cyst. Transillumination: Frontals clear, right antrum dark, left clear. Polypoid degeneration of both ethmoids. Flat tonsils, with debris. Right antrum full of pus. Left antrum contained a clear, orangecolored secretion, about half the capacity of the antrum. This fluid coagulated, no crystals, but many lymph. cells. Blood: W. B. C., 7,600; polys., 64 per cent. S. lymph., 30; L. lymph., 6 per cent. June 6, under local anesthesia, ethmoids were removed, which extended well forward and posterior and over antra. Opened sphenoids and frontals. Bone thin, membrane much thickened. Right antrum was opened under inferior turbinate. June 17, under general anesthesia, Caldwell-Luc operation was performed on left antrum. Large polypoid growth was found, also the antrum membrane was of the soft, polypoid type. Removed all membrane and growth. At the first operation, I had re-moved most of the posterior middle meatal antral wall. Extracted second bicuspid tooth, which was dead and abscessed, and removed tonsils. Present condition, 18 months later, there is no pus or polypoid membrane, no headaches

Case 3: Mrs. B. K. B., age 26 years. History: Attacks of pain in left cheek and above left eye. Posterior nasal discharge. Had had negative report from X-ray. Puncture of left antrum negative. Pain in left cheek worse for one week. Antrum punctured, much pus obtained. X-rays of teeth negative and all were vital. Examination: Nose negative. Transilluminated clear. General lavage of left antrum, via natural orifice, negative. General survey normal in every respect. X-ray of left antrum shows in its lower portion a bony shadow, projecting into antral cavity. (This may represent a dental cyst, polyp or an anomalous elevation of the alveolar portion of the maxillary bone.)

X-ray Reports, Feb. 21, 1928: V. K. Right frontal sinus is cloudy. Left frontal is clear. Right ethmoid cells are cloudy, left cells are clear. In the lateral view the entire ethmoid area looks cloudy. This would seem to indicate an involvement of all of the ethmoid cells on the right side.

Both antra are completely obscured by a cloudy infiltration, which is homogenous in character and suggests the presence of fluid in the antra. Walls of the antra show no definite evidence of thickening. Sphenoid sinus is clear.

May 21, 1929: The right frontal sinus is slightly less well aerated than the The difference in aeration between the two, however, is small. Neither

one shows thickening of the mucoperiosteal wall.

The left antrum is large and clear and shows no mucous membrane thickening. The right shows clear aeration in the upper and mesial corner. The remainder of this sinus shows a slight cloudiness. The contour of the cloudy aeration is contrasted against the clear area and is suggestive of the presence

of a large polyp within the antrum.

Ethmoidal labyrinth on the right still shows some cloudiness. The walls on the left are clear, in the lateral set of films the posterior ethmoid cells are clear, while the anterior cells are cloudy. Presumably, therefore, the cloudiness involves the anterior right cells of the ethmoid labyrinth. The sphenoid is large and clear. Turbinates are very much enlarged. The crista galli contains one large cell, which is not well aerated.

Left antrum opened through canine fossa, had a very thin wall. Polypoid growth was found occupying the lower one-third of the cavity, attached to a hard, bony ridge covering the roots of all teeth in relation to the antrum, and projecting 5 m.m. above the floor, with a narrow sulcus internally, externally and posteriorly. Result one year later, no return of pain since operation. Case 4: V. K., female, age 15 years; 1917, at 3½ years, tonsils and ade-

noids were removed for O. M. S. A. double, and suppurative cervical adenitis,

which was also opened.

June, 1927: Constant nasal obstruction and purulent discharge for past year. Sore throat several times, no headaches. Transillumination of frontals clear, right antrum dim, left dark. Nose full of mucopus, pus flowed into nose. several large drops every three minutes from under the middle turbinate. Wassermann negative. Patient put on high vitamin diet and local treatment.

Oct. 8: No pus, transilluminated clear

January, 1928: Much pus, right and left. Transillumination, frontals clear, right antrum dark. Pain and tenderness over left antrum. February: Both antra transilluminated dark, much pus. Blood chemistry normal. In May, transilluminated clear again, with only slight amount of clear mucous in nose. January, 1929: Some pus in ethmoids. Transilluminated clear. Three weeks later, much pus; on transillumination, right antrum was dark, left dim. Needle

puncture of right and left antra showed both to be full of pus. Two days later, hole was bored with Thorwald burr into both antra. March 18: Both antra negative to lavage. Now using autogenous filtrate in antra. X-ray, May, 1927, showed mass in floor of right antra. Second set were clear except for growth in floor of right antrum,

Question: How shall pansinusitis in young persons be treated?

Case 5: Mrs. J. R. M., age 55 years. History of trifacial neuralgia on left side, centering around eye, radiating to vertex and down neck. Transilluminated clear except for left antrum, which was dark. Mucopus in left side of nasopharynx. X-rays showed cloudy left antrum and unerupted canine tooth in hard palate.

Operation: Antrum entered via canine fossa, polypoid tissue found on floor of antrum. Apex of tooth projected into antrum, was exposed and lay internal to second incisor and first bicuspid. Tooth was removed and antrum drained

through inferior meatus. No more pain or discharge.

Case 6: Mrs. A. W., age 40 years. Dentigerous cyst of antrum and infection of antrum with polypoid membrane. Caldwell-Luc operation.

Case 7: Mrs. E. T., age 51 years. 1921: X-rays of sinuses and teeth negative. Hoarseness and headaches about right eye, extending to mastoid. Improved in two days by treating nasal ganglion. January, 1922: X-ray, sinus negative. December, 1928: Headaches, chronic conjunctivitis, eye fatigue, nose dry and red. Transillumination: Frontals clear, right antrum dim, left clear. On douching, right antrum was found to be full of pus, left antrum clear. X-rays: Frontals slightly cloudy. Both antra markedly cloudy, clouding. X-rays: Frontals slightly cloudy. Both antra markedly cloudy, cloudiness of right involved entire cavity. Left antrum almost completely filled with a mass of circular outline, about which the antrum was cloudy. Ethmoids moderately cloudy. Both antra were opened via the canine fossa, Right antrum contained pus and degenerated membrane, the left antrum, a large cyst filled with fluid thicker than serum. Three months later, eyes normal, no headaches, nose negative.

Case 8: R. C. S., male, age 43 years. Chronic tonsillitis with arthritis, elbows and forearm. X-ray showed growth in right antrum. Several lavages through the natural opening were negative. Centrifuged return showed only amorphous granules. Needle puncture of antrum negative. Operation: Opened right antrum via canine fossa. No growth, only soft polypoid tissue, 4 m.m. thick, on floor with thin, bony ridge across floor half-way back, 4 m.m. high. Subsequent X-ray films show part of previous shadow on external wall. Transillumination: Clear of masses, benign and malignant,

DISCUSSION.

DR. FINEMAN: Dr. Hurd has asked me to discuss the subject of the pathologic sinus from the viewpoint of the Roentgenologist. The exposure and development of a set of sinus films may be likened to the forging of a chain. Just as the weakest link measures the strength of the chain, so does the weakest step in sinus Roentgenography measure its value and usefulness as a diagnostic aid to the rhinologist.

I am a firm believer in the value of stereoscopic views in sinus work. The relationship of parts and the finer details of structures is more readily recognized. A complete examination of the nasal accessory sinuses should include at least four views; antral, ethmoid, lateral and a cephalocaudad exposure for

the separation of the right and left sphenoids.

Only recently I listened to the presentation before this Section of 10 cases of proven sphenoid disease, in none of which the routine X-ray examinations in the latreal view, revealed pathologic changes. I must point out, however, that in the lateral view, the cloudiness of one sphenoid may be largely blotted out by the superimposition of a well aerated and perhaps larger sphenoid of the opposite side. I feel fairly certain, therefore, that cephalocaudad exposures would in all probability have revealed pathologic changes in a large proportion of these cases.

Of the various sinus exposures, those made for the posteroanterior examination of the ethmoid cells are the most difficult from the technical viewpoint. A very slight rotation of the patient's head usually results in films worthless or misleading in the diagnosis of ethmoid disease. In order to recognize rotation of the head on its vertical axis, it is preferable to show most of the skull on the films. An exposure of the ethmoids with the head in the correct position will show a symmetrical relationship between the tips of the mastoids

and the rami of the mandible (diagram on board).

Now, a few words about X-ray diagnosis: In many instances in which transillumination does not reveal cloudiness of either antrum, the films will show a marked cloudiness, due to fluid or pus, or will reveal the presence of masses, polyps or polypoid thickenings of the membranes (slides). X-ray films, however, can also be misleading at times. In one case recently, the antra appeared clear on the films and yet were found to contain a great deal of pus by puncture and washing. Finally, there is the very large group of cases similar to those which Dr. Hurd has shown tonight, in which washings are negative and in which the X-ray and operative findings reveal marked disease of the mucosal lining of the antra. Oscar Hirsch, of Vienna, has recently reported 16 such proven cases of chronic catarrhal degeneration or polypoid disease of the antra.

The most important point, therefore, which I wish to emphasize tonight is that a negative antral washing does not rule out antral disease, and that the X-ray demonstration of antral changes is an indication for a most thorough and,

if necessary, exploratory examination of the antrum.

DR. M. J. MANDELBAUM: It seems that Dr. Hurd and I have had a similar experience on the question of polypoid degeneration of antrum mucosa. In the past two years. I have collected a series of personal cases numbering 20, where the antra were shown roentgenologically to definitely contain both isolated and multiple polypi. In the previous 10 years' experience it was very rare that the Roentgen report definitely suggested the presenc of antral polypi. I am told that at a recent meeting of this Section a very capable Roentgenologist made the statement that polypi could not be demonstrated Roentgenologically. Since I have become converted to "stereoscopic radiography of sinus disease" and only since then have I been convinced that stereoscopy is the only way to make a definite diagnosis of sinus changes,

Following up Dr. Hurd's remarks about the type of case where at one time there was a swelling shown in the antral mucosa, which later disappeared, or vice versa, I wonder if the condition is due to a transitory edema of the mucosa of the antrum? Not infrequently have I found during operation that the shadows diagnosed Roentgenologically as cysts or polyps consisted of extremely edematous rugae in the antral mucosa that show polyp-like shadows in the X-ray. I believe that frequently in performing a paracentesis of the antra, the needle punctures some of these edematous swellings, permits a drainage of the serum and that the swelling disappears. Subsequently, an X-ray shows no shadow and one believes that their original diagnosis has been an erroneous one.

This X-ray is illustrative of this type of case; the woman, a physician's wife, complained of right-sided neuralgic facial pains. The primary pictures taken upon X-ray plates showed no antral disease and they are excellently taken pictures. One month later, these X-ray films of the same case were taken and you could easily see a spherical mass arising from the roof of the right antrum, both in the lateral and posteroanterior exposure. A Caldwell-Luc operation showed a distinct globular mass with a sissile base exactly in the position as the X-ray showed it to be. My first thought was to curette it away, but I punctured it with the trocar and the mass collapsed. I have had eight cases similar to this one, where a puncture caused a disappearance of the cystic mass. In 20 radical cases with diagnosis of cysts or polypi of the max-illary sinus, in three cases which were subsequently X-ray, the same mass was in the same position from one to three years after the original operation, as shown in the original X-ray films.

There may be, as Dr. Coakley suggested, a constitutional idiosyncrasy or peculiar etiological factor that is the cause of this type of sinus disease. The next X-ray I show is that of "mucocele" of the left antrum, Diagnosis is considered a legitimate one, because it was cystic in nature and pressure caused an erosion of the front wall of the antrum. This also was removed through a radical Caldwell-Luc and one year after remained symptomless. The only qualification for the diagnosis of mucocele that this case did not present was that the mass did not fill the entire antrum and that it is commonly understood

that the natural drainage orifice of the antrum affected with mucocele is supposed to be blocked by the mass.

I believe Dr. Hurd shold be thanked for bringing this important type of case

before this Section.
DR. G. D. Wolf: At the last meeting of the New York State Medical Soci-I presented a number of slides illustrating the value of the injection of iodized oil in the study of antrum disease. This paper was published in the New York State Medical Journal, in the issue of Feb. 15, 1929. It seems to me that not enough stress has been laid upon the study of antrum disease, which in my limited experience has proven in many an instance to simulate the disease of any of the other paranasal sinuses. Iodized oil has heelped me in many obscure cases where the radiographic findings were not conclusive and this procedure either eliminated the antrum entirely or proved it to be the seat of the disease. This method has been so gratifying to me that I would like very much to see this iodized oil used to a larger extent in order to demonstrate whether it is worth while or not.

MYERSON: I believe that Dr. Dennis, in this country, and the Dr. M. C late Dr. Mullin, in association with him, stressed the fact that there is such a thing as latent antrum infection. They also spoke of pure polypoid changes in the antrum. It occurs to me that patients who have a pure polypoid sinusitis present themselves because of some nasal disturbance; this is usually obstruction of the passage on one or both sides. In a majority of the noses of such patients, we find very little, if anything, the matter with the other sinuses; therefore it is justifiable to perform an operation upon the antra of a radical nature in such cases. It has been my experience that these patients with stuffy noses and negative or apparently negative findings have an antrum full of polyps at operation. It has been my practice to perform a Caldwell-Luc operation upon all these cases, and the results have justified this procedure

Thanks are due to Dr. Hurd for showing his series of cases and for the interest he has aroused. Dr. Fineman is to be commended for his exceptional

and fine Roentgenological demonstration.

DR. HURD (closing): Referring to the cases Dr. Coakley spoke of: They did not seem to begin with a rhinitis. The pus poured from the nose. She is a girl of 15 years and has taken up professional dancing, and so far as we can find out there is nothing in her general condition wrong-she just fills up with pus; after three or four months, it clears up entirely.

The last case in which Dr. Fineman found a mass occupying the floor of the antrum, and which I operated on, I don't know how to account for it; it may have been a thin-walled cyst, but I got nothing in the return flow to suggest anything. I washed the antrum out, centrifuged the washing and found nothing

but some amorphous granules.

As to the question of recurrence of these polyps: They will recur if you don't take out the mucous membrane; the only way I know of is to remove

the entire mucous membrane.

As to lipiodol: When an antrum is full of polyps, I don't see how you can tell the difference between polyps and pus. Take this woman with intense pain. She had a large polypoid mass. If we had used lipiodol it would have covered all sides; it would have shown a dense shadow, and we would have seen nothing at all of the mass. We did the operation because of the intense pain.

In the last case, the man had no symptoms whatever,

A New Aid in Technical Instruction; the Use of Still Film in Place of Lantern Slides. Dr. W. Wallace Morrison.

The purpose of this communication is to bring to the attention of those members of the profession, who, up to the present time, have used the customary lantern slides for illustrating their technical lectures, or their scientific papers, etc., the possibility of the employment of still film for the projection such illustrations, and the very decided advantages of the latter method.

Still films are composed of a series of separate pictures upon standard 35 m.m. moving picture film. Each individual picture or "frame", as it is called technically, measures 18 m.m. long and 25 m.m. wide. Since each picture is so small, 50 of them may be taken upon a length of film about 3 feet long. Thus one can have 50 illustrations, enough for a long lecture or paper, upon one roll of film, which can easily be carried about in the coin pocket of one's coat.

Such a length of still film can readily be made by any amateur photographer at home with comparatively simple and inexpensive apparatus. This is accomplished, in the first instance, by the use of the miniature camera presented, and called by its makers, the "Memo" camera. It is very small, 2 inches wide, 2½ inches deep and 4 inches high, and when loaded with film weighs less than 7 ounces. It is very simply, yet well constructed. On its upper end is a telescopic range finder for ordinary photography. The face of the camera has a simple exposure counter, to indicate how many frames have been taken after the counter was set when the camera was loaded with film. The lens is an excellent one, with a very considerable focal depth, in a standard mounting. Both fixed or universal focus and focusing models are supplied. All of the models have the usual timing stops. The shutter lever is on the side of the camera. The removable back of the camera bears the simple mechanism by which the fil mis moved along from above downward for the distance required to bring an unexposed frame of film behind the lens after each exposure; it is

operated by a lever projecting from the back of the camera.

The 35-m.m. film for use with the camera is supplied in 3-foot lengths, contained in a carton measuring about 1½x1½-inch square. The film is enclosed in a reasonably light-proof wooden cartridge upon a spring roller; one end of the film projects from a felted slot for loading purposes. The camera can be loaded in subdued light, with the loss of two or three frames; the cartridge will still contain sufficient film for 50 pictures.

The pictures made by photography with this camera are, of course, negative films. For projection, positive film transparencies are printed upon standard nonburnable moving picture film, called "positive" film, either by the makers of the camera, or upon a simple printing apparatus furnished by them for the home laboratory. Such positive transparencies may be projected with any of the still film projectors on the market. The makers of the Memo camera offer a projector which they call the "Memoscope". It is inexpensive, works directly from the usual 110 volt electric supply, and with its case weighs only $4\frac{1}{2}$ pounds, so that it may be carried anywhere. It consists of a ventilated metal housing, containing one 100 watt special Mazda projection lamp, which is readily renewable. The simple condensing lens carries the light through the film, and the focusing is done by a telescopic lens mounting in front of the film, in the simplest possible manner. The film is held in the device above the lens, and moved along a frame at a time in either direction, by the handle which operates two gear wheels engaging in the sprockets in the margins of the film. When one is using this projector, the film can be moved back and forth to recall a picture once passed, with great ease and without delay, in

contrast to the projection of slides, when one wishes to reshow a slide.

To illustrate a lecture or paper, pictures in books or pamphlets, drawings, charts, tables of figures, etc. — in fact, any printed material — can be photographed by the use of the Memo camera, and the copier supplied by its makers. This consists of a pedestal upon which the camera is held by means of a standard tripod screw, fastening into the threaded socket in the base of the camera. The picture etc., to be copied, technically called the copy, is mounted upon the movable copy board and held in place by the clips provided for the purpose, after proper centering. In order to make the lens of the camera of sufficiently short focus for copying, three supplementary lenses, similar to the familiar portrait attachment, are provided with the copier. One of these is placed over the lens mounting of the camera, the copy board is moved to a given distance from the face of the camera, the copy illuminated, either with strong reflected north daylight or any bright source of artificial illumination, such as the ordinary carbon arc therapy lamp, and the picture taken by means

Illustrations can also be easily made of anatomic or gross pathologic specimens, dissections, the steps of an operation upon the cadaver, etc., since with the focusing models of the camera without the use of supplementary lenses, the subject may be approached to 3 feet or less; such closeups will give very good detail. Even greater detail can be secured by using one of the supplementary lenses of the copier, and photographing the material at the required very short distance. For instance, excellent pictures of skin lesions may be obtained by using the middle focus supplementary lens, with the skin at a distance of 83% inches from the camera face.

of a time exposure.

Stained microscopic slides may also be copied directly by a simple microphotographic method. The camera is mounted in a horizontal position on a standard triped. The slide is placed on the microscope stage, the portion to be standard tripod. The since is placed on the interescept stage, in the copied accurately focused at the required power of magnification, and the field illuminated from below by use of a strong substage illuminator. lens of the camera is then centered over the microscope ocular lens, and the picture taken. The fact that the slide is stained with the usual anilin dyes will cause the various tissues to be caught with sufficient contrast so that the picture will resemble a halftone engraving.

It is quite easy to copy Roentgen ray shadowgraphs of any portion of the body, by placing the shadowgraph against a suitable ground glass screen, before a strong source of illumination, and then photographing it as one would a picture upon the copier, by the light transmitted through the shadowgraph. Using an exactly similar method, one can copy lantern slide negatives upon still film, to replace the fragile slides with the almost indestructible film.

Note that lantern slide positives cannot be used. To add interest to a length of film of any sort of illustrations, titles and The one used by the author consists of the familiar black cloth sign board, into the slots of which removable white celluloid letters fit by means of tabs. The title is spelled out upon this board, and the whole photographed upon the copier

just as if it were a drawing.

The advantages of projection films so made over the customary lantern slides Fifty illustrations or titles on a 3-foot length of film can be carried in one's change pocket; if one has more than one length of film on the same subject, the lengths may easily be spliced together end to end, to make a single roll, 9 to 12 feet in length, with 150 to 200 pictures. Fifty lantern slides occupy a carrying case measuring 5x5x15 inches, and weighing about 10 pounds with the 50 slides. Lantern slides require a considerable space for storage if one has many; even long rolls of film can be kept in a small box in one's desk drawer. The positive films are nonburnable, difficult to tear, and unbreakable; lantern slides are exceedingly fragile. In point of cost, the advantage is overwhelmingly in favor of the film. The 3-foot length of negative film costs 50 cents; any camera store will develop it for 10 or 15 cents. The positive film transparencies are made by the camera company for \$1 for 50 pictures. Thus a 3-foot length of film for projection costs \$160 or \$1.65 for the fifty pictures; lantern slides cost at least \$1 each, and the saving by use of the film is obvious. A further advantage of the film is that, once having the negative films made, positive prints can be made at any time to replace those lost or torn; to replace lantern slides one must bear the cost as initially.

Anyone interested in amateur photography can make his own negative films; it will prove a fascinating hobby; it is done with equipment that is neither complicated nor expensive. In addition to its use in a technical capacity, the Memo camera takes excellent pictures of any subject that any other camera will take; the Memo camera has the advantages of small size and portability. Its film cartridges are so small that many can be carried in one pocket for traveling. The exposures are made simply by sighting through the finder on the camera and touching the shutter lever, as quickly as one sees a desirable The camera has been taken on long tours by those who have taken subject. thousands of pictures. From such negatives film transparencies can be made as described, for home projection. Small paper prints for the miniature album can be made from the negative direct, for a cent each. With clear negatives, paper enlargements can be made in size up to 4x5 inches at a nominal

Summary: 1. Strips of standard 35-m.m. moving picture film can be employed for the making and projection of pictures for illustrating technical lectures, etc., wiith very great advantages over the lantern slides ordinarily used. 2. Any amateur photographer can make his own films of almost any conceivable subject by using the camera and copier described. The projector offers a ready means of showing such films anywhere. 3. The camera and projector also offer a truly fascinating hobby for the amateur photographer, at home or while traveling, who wishes to record pictures of the world about him, for reviewing at his pleasure at any time.

IN MEMORIAM.

CHARLES WILLIAM RICHARDSON.

Dr. Charles W. Richardson, of Washington, D. C., was born August 22, 1861; died August 25, 1929.

He received his M.D. degree from George Washington University, Washington, D. C., 1884, and also received the degree of M.D. from the University of Pennsylvania in 1884.

For over thirty-five years he was engaged in Washington in the practice of otolaryngology and interested himself in many of the civic and medical activities of his home city. He enjoyed the unusual distinction and honor of serving as president of each of the several national otolaryngological societies with which he had been identified for so many years. Thus he served as president of the American Laryngological Association, the American Climatological Association and the American Laryngological, Rhinological and Otological Society. He was Chairman of the Section of Laryngology of the American Medical Association and also a member of the Board of Trustees of that body. He was a Fellow of the American College of Surgeons and Professor Emeritus of Laryngology and Rhinology at George Washington University. He was a member of the American Otological Society and of the American Academy of Ophthalmology and Otolaryngology.

For many years he was the Delegate of the American Laryngological Association, the American Otological Society, the American Congress of Physicians and also served as a member of the House of Delegates of the American Medical Association.

During the World War, he was named Director of the Department of Reconstruction for Deaf and Defective Speech Soldiers and directed the organization of the special hospital for this service at Cape May.

He was Chairman of the Committee on the Deaf Child of the American Medical Association. About five years ago he succeeded in interesting the National Research Council in financing a survey of Schools for the Deaf and, with the assistance of a group of otologists, instituted the examination of several hundred thousand school children throughout the country in order to determine the percentage and character of defects in hearing that only such a survey could bring about.

He was a frequent contributor to the literature of otolaryngology and wrote special sections in the field for several general systems of medicine.

His home, in Washington, was the center of many auspicious and important social gatherings to which his otolaryngologic and general medical confreres were always graciously welcomed.

He was a prominent figure at all meetings of the various otolaryngological societies and his presence and council will be sadly missed by the colleagues with whom he had been so closely identified for the past three decades and more.

To his devoted wife and daughter we offer our sincere sympathy for we knew of the close ties that existed there. M. A. G.

VITTORIO GRAZZI.

Vittorio Grazzi, the last of Italy's quartette of distinguished pioneer laryngologists, which included Gradenigo, Ferreri and Massei, died April 17, 1929.

He was born Aug. 2, 1849, at Sinalunga, graduated with honors at the University of Sienna, qualifying after some years of internship and special scholarships in the practice of otolaryngology.

He was professor ordinarius of laryngology at Pisa and Florence, and his lectures and clinics were always popular with students because of the simplicity, clarity and grace with which he presented his subject matter.

He was especially interested in the problem of congenital deafness and the schools for deaf children in Italy; in the development of bronchoscopy; the treatment of laryngeal tuberculosis and many other otolaryngological topics of wide communal interest.

He founded the monthly publication, "Bolletino delle Malattie dell' Orecchio della Gola e del Naso", now in its forty-seventh (XLVII) volume, a special journal in which many of his best contributions to otolaryngologic literature were published.

When the Fourth International Otological Congress met in Florence in 1895, he was its presiding officer and presented his address in English. To those of us who had the privilege of knowing him he always radiated good fellowship and gentle courtesy. He was keenly alert and interested in the progress of otolaryngology, was a frequent visitor to the best clinics of Europe, a member of important special societies and a familiar and esteemed personality at all international congresses of otology and laryngology.

It was characteristic of his generous spirit and kindly nature that he should leave in his testament substantial bequests to the several institutions with which he was actively associated for many years.

To the Medico-Physical Academy of Florence, he left 20,000 lira, requesting that the interest accumulate for five years, this sum to be expended for a prize, to be known as the Vittorio Grazzi prize, for the best work in either Latin or Italian on the embryology, anatomy or physiology of the organs of hearing and their accessories.

To the University of Pisa, where he was active for many years as professor ordinarius, teaching otolaryngology, he left a fund of 25,000 lira, the interest of said sum to accumulate for three-year periods and this amount to be given in each instance for a Vittorio Grazzi prize for the best work in Italian in otolaryngology.

To the University of Florence he left his library, armamentarium and interesting collection of photographs and autographs of distinguished colleagues.

Smaller bequests were made to the Royal Institute for Deaf-Mutes, in Sienna; the Royal Institute of Music, in Florence; and the Institute Gualendi, with all of which he was long and actively identified.

His contributions to otolaryngological literature number over 150 articles, all presented in charming style, enthusiastic, sincere and timely.

We say with him: "Labor improbus omnia vincit."

M. A. G.

